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16. ABSTRACT A review of the 1981 world literature was conducted on the effects of pollution on freshwater fish. This review contained 353 references and included papers on water quality such as dissolved gases and pH; chemical pollutants such as insecticides, herbicides, piscicides and anesthetics; inorganics, organics and industrial and municipal effluents including chlorine and wastewater and pulp and paper. Papers from reviews and symposia and those dealing with physical factors were also included. Within the above sections, papers were organized into general articles, surveys, toxicology, bioaccumulation, physiology and behavior.					
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Effects of pollution on freshwater fish

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REVIEWS AND SYMPOSIA

Swain and Shannon¹ compiled and edited a joint U. S.-USSR scientific symposium on the theoretical aspects of aquatic toxicology. Areas of research discussed included organophosphate and chlorinated hydrocarbon toxicity to fish and other aquatic life, water quality standard setting, monitoring strategy, and toxicity prediction. A symposium on aquatic toxicology was sponsored by the American Society for Testing and Materials.² Areas of discussion included toxicity tests on inorganic and organic chemicals as well as effluent testing, bioconcentration, and testing methods for fish and other aquatic organisms. The literature concerning procedures and results of bioassay testing of fish and other organisms was reviewed by Maciorowski *et al.*³ Results of toxicity tests on water quality parameters, inorganic and organic chemicals as well as some industrial pollutants were found in the literature and a comparison of various test methods were discussed. A compilation of the toxicity of chemicals to fish and aquatic invertebrates was reported by Johnson and Finley.⁴ Results included the effects of approximately 260 chemicals including metals and organic chemicals to several fish and invertebrate species tested at the Columbia National Fisheries Research Laboratory in Columbia, Missouri, from 1965 to 1978. Lists of species used and chemicals included were separately listed and then described with the toxicity information. The effects of pollution on freshwater fish from around the world were reviewed by Spehar *et al.*⁵ Russom⁶ compiled a bibliography of all publications completed or sponsored by the U. S. Environmental Protection Agency (EPA), Environmental Research Laboratory-Duluth for the years 1976-1978. Included

were journal articles and EPA research reports. Many specialty areas were covered but most dealt with aquatic toxicology and environmental effects of chemicals on fish and other aquatic life. Johnson and Bailey⁷ developed a computerized system to rate chemical classes as to their effects on fish reproduction. The report presented several systems for rating inorganic and organic chemicals by structure and test types. Tables were presented showing toxicity concentrations for inorganic and organic chemicals and depicting the rating of comparative toxicity by the various systems.

A review of the literature on mixtures of toxicants on European freshwater fish was prepared by the European Inland Fisheries Advisory Commission.⁸ The toxicity and accumulation of metals, organic chemicals, chlorine, and other disinfection agents to fish as well as invertebrates and plants were discussed. Murphy⁹ reviewed the literature on toxicity and bioaccumulation of heavy metals in the environment. Data presented included information on several fish species as well as on other aquatic organisms. Jenkins^{10,11} reviewed the literature on biomonitoring of trace metals in the environment in two volumes. Volume 1 included analysis of the comprehensive data in Volume 2 on bioaccumulation and bioconcentration of 14 trace metals in plants and animals including fish. Data were analyzed on the use of plants and animals for monitoring environmental gradients of trace metals and on biomagnification of trace metals in food chains.

The literature on the toxicity of organophosphorus pesticides to fish was reviewed by Cizek and Svobodova.¹² The authors discussed the physiological and toxicological effects of these compounds on fish and their use in fish culture. Schneider¹³ published a toxicological evaluation of pesticides in relation to their effects on mammals and aquatic life, including several species of fish. The handbook contained data on approximately 1 000 aquatic tests and LC50 (concentration lethal for 50% of animals tested) data for the 9 years prior to publication. Appendixes containing test methods and chemical information were also included.

Connell and Miller^{14,15} reviewed the literature on the behavior and effects of sublethal concentrations of petroleum hydrocarbons in aquatic systems in a two-part review. Information included sources, dispersion, physical, and environmental effects. Only limited information was presented on freshwater fish. A literature survey on petroleum hydrocarbons in the aquatic environment was published by Jones *et al.*¹⁶ and included papers dealing with sources, accumulation, toxic effects, and fate of several kinds of chemicals including some affecting fish. McKendrick *et al.*¹⁷ reviewed the literature concerning the effects of oil spills in cold climates. It was found that only a limited amount of studies on the effects of oils to fish and other aquatic life have been done. For crude oil, 96-hour LC50 values ranged from 2.75 mg/l of total aromatic hydrocarbons for Dolly Varden (*Salvelinus malma*) to 10.45 mg/l for the threespined stickleback (*Gasterosteus aculeatus*). A review on the presence of unusual polyhalogenated chemical residues in fish tissue was conducted by Kuehl.¹⁸ Information included sources, transport, and accumulation of these chemicals. The review showed that there exists a large need for continued chemical-residue characterization and methods development work.

An annotated bibliography on the effects of pH on fish was prepared by Daye.¹⁹ Another review on the impact of low pH

on fish and fish populations was reported by Fritz.²⁰ Laboratory experiments showed that toxic effects of low pH differed among species, populations, and age groups of the same species. Field studies indicated that toxicity from low pH was compounded by other water characteristics. Several other effects on fish caused by low pH were discussed. Lynch and Corbett²¹ presented a short review of the situation regarding acid precipitation and its effect on aquatic ecosystems. Acidic fallout was indicated as the cause of the elimination of fish and other aquatic life in many aquatic areas especially in Scandinavia, Canada, parts of Europe, and in the northeastern U. S. The authors concluded that efforts must be made immediately to protect these countries' valuable forest and aquatic resources. The effects of acid precipitation on Pennsylvania waters were surveyed by Arnold *et al.*²² Thirty-four percent of the analyses showed that lakes and streams have decreased in pH, alkalinity or both over a period of 1 year. In addition, fish-counting data showed that the number of fish species present in these waters decreased 58% in 40 out of the 107 analyses made.

Coad²³ published a general overview of the factors affecting the distribution and abundance of freshwater fishes in Iran. These included climate, devegetation, irrigation, and natural water level fluctuations. In addition, factors related to industrialization and population increases were described. The toxicity of landfill leachates were both reviewed and studied, using static bioassays or the residual oxygen bioassay techniques with rainbow trout (*Salmo gairdneri*).²⁴ It was concluded that natural leachates were highly toxic within the confines of landfills and also at the point of receiving stream discharge, that water quality parameters such as pH and temperature changes were important toxicity factors, and that physical pretreatment and leachate recycling were both shown to be effective in reducing leachate toxicity. Hall and Knight²⁵ reviewed the literature on abundance studies of salmonids with special emphasis on relationship to non-point pollution. The authors included recommendations for the use of this information in designing studies to estimate impacts of such sources. Walden and Howard²⁶ presented a review on the toxicity of pulp and paper effluents on fish. Acute and sublethal toxicity as well as the toxic constituents and detoxification processes of these effluents were discussed. Issues concerning the potential impact of lignite surface mining on fish and wildlife were reviewed by Haynes.²⁷ The review included considerations of habitat destruction and inadequate reclamation strategies. Protection factors, data acquisition, and assessment needs were discussed. Weitkamp and Katz²⁸ reviewed the literature on the effects of supersaturation on fish and other aquatic life. The causes and cures of gas bubble disease, the main damaging effect of supersaturation, were discussed.

WATER QUALITY

Dissolved gases. Montgomery and Becker²⁹ reported on gas bubble disease in smallmouth bass (*Micropterus dolomieu*) and northern squawfish (*Ptychocheilus oregonensis*) collected from the lower Columbia and Snake Rivers. Emboli were found in 72% of the bass and in 84% of the squawfish. Hemorrhages were noted in the caudal, anal, and pectoral fins of some bass. The presence of gas bubble disease corresponded to high gas supersaturation in the river during spring runoff.

The effects of gas supersaturation on the warmwater fishery of the upper Osage River in Missouri in 1978 were described by Crunkilton *et al.*³⁰ Levels up to 139% caused nearly one-half million fish deaths with nitrogen being the primary gas responsible and high temperatures seemed to heighten the effect. The responses of Atlantic croakers (*M. undulatus*) to supersaturation by nitrogen and oxygen with temperature variation were studied by Chamberlain *et al.*³¹ Mortality and vertical movements of fish were analyzed and a response model with its physiological basis was prepared. Nebeker *et al.*³² compared the responses of speckled dace (*Rhinichthys osculus*) and cutthroat trout (*Salmo clarki*) exposed to air supersaturated water. Speckled dace were found to be more tolerant than cutthroat trout. Signs of the disease were exhibited by both species and were similar to those seen with other salmonids examined in earlier studies. Stevens *et al.*³³ reported that coho (*Oncorhynchus kisutch*), sockeye (*Oncorhynchus nerka*), and chinook (*Oncorhynchus tshawytscha*) salmon smolts and rainbow trout (*Salmo gairdneri*) were found to avoid supersaturated water at levels of 145 and 125%. However, steelhead (*Salmo gairdneri*) did not consistently avoid this water and sometimes died from gas bubble disease. Other experiments³⁴ showed that steelhead trout exposed to 120 to 140% of normal air pressure at various depths survived best at the deepest levels. The significant measure of supersaturation seemed to be the pressure of dissolved gases in excess of the sum of barometric and hydrostatic pressure. Hill *et al.*³⁵ found that when dissolved oxygen (DO), pH, and total dissolved solids were presented to striped bass (*Morone saxatilis*) as single gradients, the fish selected a preferred zone. The preferred zone for a single parameter was often altered when two or all factors were presented simultaneously. Of the three parameters tested, pH was more influential than DO and dissolved solids in affecting the locomotor responses. In a study by Matthews and Styron,³⁶ a headwater cyprinid (*Phoxinus phoxinus*) and darter (*Etheostoma flabellare*) of the Roanoke River drainage area was compared to three mainstream cyprinids (*Notropis ardens*, *N. albeolus*, and *N. cerasinus*) and two darters (*E. podostemone* and *Percina roanoka*) for tolerance to abrupt changes in DO, temperature, and pH. The headwater fish were found to be more tolerant of low O₂ conditions than those in the mainstream.

Metabolic rates of *Tilapia mossambica* exposed to O₂ levels below saturation and during a subsequent recovery period were measured by Peer and Kutty.³⁷ Oxygen consumption at 30° and 35°C decreased with decreased ambient O₂ during the hypoxic phase. The recovery metabolism indicated that this fish accumulated an O₂ debt at 30°C but not at 35°C. The changes in metabolic mechanisms due to hypoxia as affected by temperature were discussed. Channel catfish (*Ictalurus punctatus*) were exposed to sublethal hypoxia for 24, 48, or 72 hours and hematological factors were analyzed by Scott and Rogers.³⁸ Hemoglobin, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, plasma lactic acid, and plasma glucose values showed significant deviation from controls, whereas, no changes were found for hematocrit, total plasma protein, RBC, WBC, mean corpuscular volume and differential WBC. Plasma corticosteroid concentrations in channel catfish increased from 1 to 5.9 µg/100 ml when exposed to 0.1 mg O₂/l.³⁹ On restoring O₂, the values returned to normal within 30 minutes, although a secondary increase

occurred 6 hours later. Steroid levels increased in fish exposed to a concentration of 0.2 mg O₂/l for 3 days. The effects of hypoxia on hemoglobins and ATP levels in *Cyprinus carpio*, *Sarotherodon mossambicus*, and rainbow trout were discussed by Smit and Hattingh.⁴⁰ Marked interspecies differences were noted. ATP concentrations declined with a reduced O₂ content of the water for all species. Chemical stress on rainbow trout caused by hypoxia, ammonia, and phenol was measured by Swift⁴¹ in terms of changes in blood parameters. The factors measured were packed cell volume, glucose, cortisol, chloride at different regimes of toxicant concentration and temperature. Plasma cortisol provided the best estimate of stress. The growth rate and food conversion efficiency of coho and sockeye salmon were determined at DO concentrations ranging from 2 to 15 mg/l at 15°C.⁴² It was concluded that above a critical level ranging from 4.0 to 4.5 mg/l, both measures were not limited when tested for relatively short periods of 6 to 8 weeks. Slightly higher hematocrits in fish exposed to lower O₂ levels indicated that adaptive mechanisms for improved respiratory capacity at subcritical O₂ concentrations may exist.

Paller and Heidinger⁴³ described the mechanisms of delayed ozone toxicity to bluegills (*Lepomis macrochirus*). An exposure concentration of 0.09 mg/l caused the destruction and loss of epithelial cells covering the gill filaments. Extensive fungal infections often followed exposure to ozone resulting in the death of the fish. An abrupt decrease in serum osmolality was detected in exposed bluegills leading to mortality when the value decreased below 50 mOsm. The toxicity of ozone-producing oxidants to striped bass eggs, larvae, and fingerlings was studied by Hall *et al.*⁴⁴ and compared to previously published chlorine toxicity data. Eggs were most sensitive to the oxidants in freshwater than in estuarine water. The toxicity of ozone and chlorine was found to be similar with eggs and larvae in estuarine water.

Short-term bioassays were conducted by Ruffier *et al.*⁴⁵ to evaluate the acceptability of the EPA water quality criteria for un-ionized ammonia. The LC50 values from this study were consistent with values in the literature and exceeded the value currently recommended by the EPA. Tests on the effects of fluctuating exposures of ammonia to rainbow trout and cutthroat trout ranging from 96 hours to 4 weeks⁴⁶ showed that fish could withstand constant concentrations better than fluctuating concentrations and could tolerate short-term concentrations slightly above the acutely lethal level. Pulsing the dosages caused acclimation toward subsequent exposures. Larger rainbow trout were found to be more vulnerable to acutely toxic concentrations than smaller trout. Other tests⁴⁷ showed that when rainbow trout were exposed to aqueous ammonia at reduced DO concentrations, there was a positive correlation between the LC50 for un-ionized ammonia and DO over the range tested (2.6 to 8.6 mg/l); ammonia toxicity increased as DO decreased. Better correlations with DO were found in shorter exposures than longer ones. Rice and Bailey⁴⁸ exposed eggs and alevins of *Oncorhynchus gorbuscha* to ammonia in short- (96-hour) and long-term (up to 61-day) tests. Late alevins near emergence were found to be the most sensitive life stage. Concentrations as low as 1.2 mg/l in 61-day tests reduced the growth of fry. Concentrations at 10 mg/l of ammonia stimulated early emergence of immature fry.

The toxic mechanism of action of ammonia on rainbow trout exposed for 4 to 48 hours was studied by Arillo *et al.*^{49,50} Brain

levels of glutamate, GABA, total sugars, ATP, NADH, and succinate decreased while tissue- NH_3 , glutamine and lactate increased. Liver levels of total sugars, succinate and lactate decreased, while tissue- NH_3 , glutamine, and the taurine/glycine ratio increased. Gill sialic acid increased then decreased slightly. In another study,⁵¹ renal renin activity increased in rainbow trout exposed to ammonia, with effects being proportional to the log of the ammonia concentration. These data contributed new information concerning renin activity and diuresis in freshwater fish. The effects of ammonia on the metabolism of nine carp species were examined by Shaffi.⁵² An inverse relationship was recorded between the concentration of ammonia and the fall in pH and DO in the water. With ammoniacal nitrogen and CO_2 , the relationship was reversed. A direct relationship was recorded between ammonia concentration and serum glucose and lactate but ammonium concentration was inversely proportional to the fall in tissue glycogen. The effects of abrupt sublethal gradients of ammonium chloride on the activity level, turning, and preference-avoidance behavior of bluegills were monitored by Lubinski *et al.*⁵³ Bluegills exposed to 4.0 $\mu\text{g/l}$ of un-ionized ammonia tended to show a slight increase in activity, a slight increase in turn sizes and an initial exploratory response where fish positioned themselves at or near the gradient. No avoidance behavior was conclusively established.

The effects of HCN on embryonic, juvenile, and adult American flagfish (*Jordanella floridae*) were described by Cheng and Ruby.⁵⁴ Results of exposures of 0.065 to 0.15 mg/l showed that the embryonic stage was especially sensitive and that these concentrations caused reductions in hatching and fry survival. Dixon and Leduc⁵⁵ found that CN affected growth and metabolic rate, and caused degenerative hepatic necrosis in juvenile rainbow trout in 18-day exposures at concentrations ranging from 0.01 to 0.03 mg/l . Torrains⁵⁶ discussed the acute toxicity of H_2S to fish during harvesting operations of commercial catfish farms, which included a discussion of causes, prevention, and cure. The concentrations ranged from 1.2 to 18.1 mg/l . Cytochrome oxidase levels in the tissue of exposed fish were monitored and found to be inhibited by relatively low dosages of H_2S .

pH and acid stress. Kennedy⁵⁷ evaluated the effects of reduced pH on reproduction and recruitment in a population of lake trout (*Salvelinus namaycush*) from an experimentally acidified lake (mean summer epilimnion pH of 5.84) and compared the results with fish results from a control lake. Acid-stressed fish showed increased embryonic mortality, morbidity, and embryonic malformations compared to control fish. The author suggested that there was a critical pH (~ 6.0) for lake trout reproduction and survival based on results of survey reports. The effects of experimentally increased acidity on the ecology of a stream in the Hubbard Brook experimental forest, New Hampshire, were determined by Hall *et al.*⁵⁸ At pH 4, water concentrations of several metals along with the drift of several species of insects were increased. Emergence of some insects were decreased. Brook trout (*Salvelinus fontinalis*) avoided the lower pH but those exposed continuously to pH 4 to 5 showed no evidence of pathological changes in gill structure. Gunn and Keller⁵⁹ described the effects of limestone on the survival of eggs and fry of rainbow trout in an acid lake. Incubation of eyed rainbow trout eggs in limestone enhanced hatching success and sac fry survival. Comparison of the ef-

fects of limestone and noncalcareous gravel in sheltered and wind-exposed substrates on trout survival were studied. The use of limestone as a tool in maintaining salmonid populations in acid lakes was also discussed. Some effects of the acidification of Atlantic salmon rivers in Nova Scotia were described by Farmer *et al.*⁶⁰ The treatment of the Mersey River, which supplies an Atlantic salmon hatchery, with calcium carbonate helped to significantly increase the survival of Atlantic salmon (*Salmo salar*) parr. Annual mortality at the hatchery prior to treatment was attributed to the soft acidic water. The chemical characteristics of several rivers on the Atlantic side of mainland Nova Scotia were determined and the data related to that from 25 years earlier and to salmon populations were discussed. Growth responses of yellow perch (*Perca flavescens*) to acidification of mountain lakes in Ontario was studied by Ryan and Harvey.⁶¹ Young yellow perch responded with increased rates of growth while older fish responded with reduced growth rates. The authors suggested that the growth rate of yellow perch may serve as an indication of acid stress.

The effect of pH on Na^+ uptake in Norwegian brown trout (*Salmo trutta*) from an acid river (Tovdal River, Great Britain) were measured by McWilliams.⁶² Sodium ion uptake by brown trout was dependent on external acidity, with wild fish being more tolerant than hatchery fish. The inability to regulate ionic balance was an important factor in fish mortality from acid water. In another study,⁶³ it was reported that the ability of brown trout to acclimate to acidic conditions (pH 6.0) was dependent largely on changes in gill permeability to Na^+ . Brown⁶⁴ tested the effects of various cations on the survival of brown trout exposed to low pH (3.5 to 4.0). Calcium prolonged the survival time as did Na^{2+} and a mixture of Al and Na^{2+} . Potassium and Mg^{2+} had no effect. The previous history of the fish had an effect on their sensitivity to low pH. The effects of cations and pH on the motility of spermatozoa of rainbow trout were examined by Baynes *et al.*⁶⁵ Potassium ion and a pH less than 7.8 were confirmed as being inhibitory to the activation of spermatozoa. Sodium ion was a mild antagonist to the inhibition, whereas both Ca^{2+} and Mg^{2+} were strongly antagonistic. McDonald *et al.*⁶⁶ determined that the physiological responses of 1- to 2-year-old rainbow trout exposed to low pH were dependent on Ca^{2+} concentration. High Ca^{2+} levels protected against low pH (3.0 to 3.2) but low Ca^{2+} increased survival at higher pH (3.8 to 4.0). Iono-regulatory failure was found to be the primary cause of death in fish exposed to low pH in soft water.

Seven-day tests conducted by Graham and Wood⁶⁷ were used to establish the influence of water hardness (140 versus 14 mg/l CaCO_3), acid type (HCl versus H_2SO_4), and fish activity (rest versus exercise) on acid toxicity to fingerling rainbow trout. The pH LC50 ranged from 4.1 to 4.5; hardness reduced acid toxicity and exercise generally potentiated toxicity. Physiological mechanisms and ecological significance were discussed. Carrick⁶⁸ studied the relationship between O_2 consumption and pH of brown trout (*Salmo trutta*) fry. No significant differences in O_2 uptake were found at the two pH levels studied (pH 5 and 7).

The effect of acute acid stress on isolated perfuse gills of rainbow trout was determined by Jackson and Fromm.⁶⁹ Lowering the pH from 7.2 to 3.5 had no significant effect on the transfer capacity of gills perfused with or without epinephrine. The vascular resistance increased with gills not treated with

epinephrine, but the gills showed no vasomotor response if treated with epinephrine. The pH of the efferent perfusate was not affected by the acid stress. McDonald and Wood⁷⁰ studied the effects of low pH on branchial and renal acid and ion fluxes in rainbow trout. At a pH of 4.2, there was a branchial influx of acid, provoking a progressive depression in blood pH and HCO_3^- , and a marked increase in renal acid excretion. Low pH exposure also impaired gill ion regulation. An experiment concerned with acid-base and electrolyte status in the blood of carp (*Cyprinus carpio*) exposed to various levels of low pH⁷¹ showed that at pH 5.1, the parameters were unchanged, but the base loss was increased by a factor of 2. At lower pH values, the base loss was increased and the Na^+ and Cl^- concentrations fell. At a pH of 3.5, tissue hypoxia appeared as an additional factor aggravating the acid-base and electrolyte status. Rainbow trout were used in another study⁷² to measure ventilatory and arterial acid-base parameters on exposure to acidified tap water (pH 4.0) during 2-hour, 5-day, and 12-day time periods. There was no short-term (2-hour) response to an increase in H^+ alone. During the 5- to 12-day treatment there was electrolyte loss, metabolic acidemia, loss of total CO_2 , and an increase in erythrocyte concentration. The ability to compensate acidemia and loss of total CO_2 when the ambient HCO_3^- was increased 10-fold at a constant pH of 4.0 suggested that a system exists in the fish for the active uptake of environmental HCO_3^- . A morphometric study was carried out by Zuchelkowski *et al.*⁷³ to analyze effects of acid stress (pH 5.7 to 6.2) on epidermal mucous cells of brown bullhead (*Ictalurus nebulosus*). Mucous cell volume density was increased after a 5-day exposure, although there was no change in cell diameter or cell volume. However, the number of mucous cells was nearly twice that of controls. The results indicated that the acid stress caused hyperplasia without hypertrophy.

The effect of low pH on chorionase activity of Atlantic salmon eggs was investigated by Haya and Waiwood.⁷⁴ After analysis, it was concluded that there seemed to be at least two reasons for a reduced hatching success of salmon eggs exposed to low pH: a decrease in chorionase activity and a change in the physical structure of the outer layer of the chorion. Lee and Gerking⁷⁵ found that eggs of the desert pupfish (*Cyprinodon neradensis*) were susceptible to acid exposure immediately after fertilization until the membranes water hardened. A comparison of these results was made with previous work and the effects of acid conditions on oogenesis were discussed.

CHEMICAL POLLUTANTS

Insecticides. Schmitt *et al.*⁷⁶ analyzed fish from 100 stations each year from 1970 to 1974 for organochlorine residues as a part of the National Pesticide Monitoring Program by the U. S. Fish & Wildlife Service. DDT and its metabolites declined nationally during those years, but remained widespread with concentrations continuing to be high in areas where DDT was used extensively. Dieldrin and endrin residues were relatively unchanged while toxaphene occurrence increased. Hunter *et al.*⁷⁷ examined the filets of 99 fish (11 species) from Lake Texoma, Texas and Oklahoma, for residues of organochlorine compounds and also attempted to determine the relationship of the residues in the fish to trophic level and geographical location. PCBs and p,p'-DDE were detected in most

species at levels up to 1 000 ng/g and 127 ng/g, respectively. Heptachlor, p,p'-TDE, and o,p'-DDT were present in detritivores and herbivores. Chlordane was found in all trophic levels while dieldrin and p,p'-DDT were found only in detritivores and carnivores. Water, bottom sediments, and fish samples from the Atchafalaya Basin in Louisiana were analyzed for 9 organophosphorus compounds and 18 organochlorine compounds by Hern *et al.*⁷⁸ Organophosphorus pesticides were not detected. Only PCBs were found in water, bottom sediments, and fish. Aldrin, dieldrin, and DDT and its derivatives were detected in bottom sediments and fish. Chlordane was found only in bottom sediments. Chlorinated pesticide levels in the Po River and Adige River, Italy, were determined for 1977 through 1978 by Galassi and Provini.⁷⁹ Aldrin, heptachlor, heptachlor epoxide, endrin, HCB, and p,p'-DDE were not detected. Levels of α BHC, γ BHC, and p,p'-DDT were lower than those reported for 1971 through 1973. Watanabe *et al.*⁸⁰ measured residues of the insecticides BHC and DDT and the herbicide CNP in isaza (*Chaenogobius isaza*) from Lake Biwa, Japan. In all samples collected from 1966 to 1976, BHC (α , β , γ , and δ -isomers), DDT and its derivatives (DDE and DDD), and CNP were detected. The highest concentrations measured were 2.09 $\mu\text{g/g}$ for total BHC, 0.89 $\mu\text{g/g}$ for total DDT, and 0.11 $\mu\text{g/g}$ for CNP. Although the use of BHC and DDT was stopped in 1971, the concentration of these two pesticides have not shown much decrease. Horii *et al.*⁸¹ identified γ -chlordene in the freshwater fish, *Zacco platypus*, collected from the Tama River in Japan. The authors indicated that this compound was a common contaminant of aquatic animals in the area. Several other organochlorine compounds also were found. Organochlorine residues in four lakes in the Kenya Rift Valley were studied by Lincer *et al.*⁸² DDE levels were found to be highest, 2.13 mg/l, in the bottom-feeding fish, *Labeo cylindricus*. El Zorgani⁸³ measured residues of DDT in fish from the Gezira canals and Lake Nubia, Sudan. All muscle samples of fish from the Gezira canal contained p,p'-DDT, and its breakdown products DDE and TDE. Of the 58 samples from Lake Nubia, p,p'-DDE was found in 10 samples but p,p'-DDT was found in only 3 muscle samples. Total DDT in muscle ranged from 23 to 184 $\mu\text{g/kg}$. The impact of thermal discharge from a nuclear power plant on residue levels of DDT and PCB in perch (*Perca fluviatilis*) and roach (*Leuciscus rutilus*) was investigated by Edgren *et al.*⁸⁴ The monthly mean values for DDT and PCB were significantly higher in perch from the heated area than in perch from the intake area. No significant differences could be established for the roach.

The effects of an aerial application of carbaryl on brook trout (*Salvelinus fontinalis*) were reported by Haines.⁸⁵ The *in situ* toxicity tests indicated no mortality associated with the insecticide. The brain AChE activity of trout 4 and 8 days after the first treatment was significantly lower than that of control fish. However, this activity was normal after 9 days. There was no indication that the pesticide application affected the condition factor of the trout. Morrison and Wells⁸⁶ conducted a comprehensive study of the effects and fate of fenitrothion in a stream environment following aerial spraying of a Scottish forest. There was no evidence that the resident fish nor caged brown trout (*Salmo trutta*) and Atlantic salmon (*Salmo salar*) were disturbed by the treatment, which reached a maximum concentration of 18 $\mu\text{g/l}$ in 1 hour. The rate of uptake of fenitrothion in the skin, stomach, and muscle of the

caged fish followed closely the rise in water concentration, and the rate of loss was almost as rapid as that in the water. Field studies to determine the long-term effects of years of intensive pesticide application on aquatic life in orchard drainage ditches were conducted by Heckman.⁸⁷ These results were compared to a similar study made 25 years earlier. Comparison of the community structures and abundance of the various species indicated that many species have become resistant while other species have been eliminated. No direct effects of the pesticides on fish were suspected. The hematological characteristics of fish exposed to six sequential aerial sprayings (July through October 1978) of endosulfan for tsetse fly control in Botswana were examined by Matthiessen.⁸⁸ In all cases, blood cell counts were significantly elevated during spraying and plasma protein levels were often disturbed. Total hemoglobin did not change, but the mean weight of hemoglobin per erythrocyte declined in *Tilapia* sp. In most cases these blood characteristics began returning to normal before spraying stopped and reached pre-spraying levels within 6 months. Sanders *et al.*⁸⁹ evaluated the effects of abate on bluegills (*Lepomis macrochirus*) and invertebrates in small ponds treated at a concentration of 4 µg/l to simulate the normal application rate and at a concentration of 40 µg/l to simulate an overspray or spill. In the ponds treated at 4 µg/l, fewer bluegill fry were produced than in the control ponds. However, these fry and the adults grew faster probably as a result of heavy feeding on dead or moribund dipteran larvae. In the ponds receiving 40 µg/l, bluegills had an accelerated development of ovaries, but the number of fry per female was reduced. Brain AChE was strongly inhibited. No effect on growth of bluegill fry was found. Mulla *et al.*⁹⁰ studied the impact of weekly applications of decamethrin and permethrin on mosquitofish (*Gambusia affinis*) and desert pupfish (*Cyprinodon maculatus*) in experimental ponds. At the mosquito larvicidal rate (0.4 µg/l for decamethrin and 9 µg/l for permethrin) and at five times this concentration, there was no adverse effect on survival and reproduction of either species. A field study to determine the efficacy of SIR 8514, insect growth regulator, on pest diptera and on non-target aquatic life in a pond was conducted by Colwell and Schaefer.⁹¹ Bluegills and mosquitofish accumulated maximum concentrations of 0.4 µg/g at 2 days post-treatment. Residues in fish were not detected at 28 days post-treatment. No fish mortality was observed. Allen *et al.*⁹² determined the persistence of isobornyl thiocyanacetate (Thanite) in treated ponds, identified a major metabolite of the insecticide in largemouth bass (*Micropterus salmoides*) and carp (*Cyprinus carpio*), and studied the elimination of the metabolite. Analysis for residues in the fish exposed to 1 mg/l of Thanite revealed only trace amounts of the parent compound. A metabolite, isobornyl α-(methylthio)acetate, was isolated and identified. Residues of this metabolite were eliminated rapidly after the fish were returned to clean water.

Autopsy techniques for investigating pesticide poisoning in fish were discussed by Braun *et al.*⁹³ The authors concluded that there was only a small possibility to prove pesticide poisoning by histopathological or biochemical analysis. On the other hand, they suggested that pesticide residues in the organs of fish indicated pesticide toxicity. Residue levels of endosulfan, lindane, and 2,4,5-T-ester in fish caused by acute and chronic exposure were given. Tomizawa⁹⁴ re-evaluated rice

field pesticides using a modification of the model ecosystem proposed by Metcalf. The guppy (*Lebistes reticulatus*) was used as the test fish. It was concluded that the persistence and distribution of rice field pesticides in the model ecosystem depended on their chemical structure and that the model ecosystem may be applicable and useful for predicting the environmental fate of these pesticides. A six-compartment equilibrium model and a three-compartment kinetic model to evaluate the hazard to aquatic life (including several species of fish) of pesticides from forest spraying was used by Zitko and McLeese.⁹⁵ The models to estimate environmental concentrations were based on physico-chemical properties of the pesticides and on application rates. Estimated environmental concentrations and LC50 values and bioconcentration factors (BCFs) were compared with those of fenitrothion. Estimated environmental effects of other pesticides were evaluated relative to the known environmental effects of fenitrothion. Schnoor⁹⁶ developed a model of pesticide fate and bioconcentration to assess the long-term effects of dieldrin in Coralville Reservoir, Iowa. This model was calibrated with field data from 1968 to 1978. Dieldrin residues in fish, sediment, and water were found to be declining at about 15% per year. Approximately 50% of the pesticide load was exported from the reservoir in the outflow. Model projections indicated that by 1986 the dieldrin residues in the flesh of bottom-feeding fish should average less than 100 µg/kg.

The effect of some experimental variables on the acute toxicity of five insecticides to cutthroat trout (*Salmo clarki*), a stonefly, and an amphipod were determined by Woodward and Mauck.⁹⁷ The 96-hour LC50 values of the technical grade insecticides to cutthroat trout ranged from 0.38 mg/l for trichlorfon to >100 mg/l for acephate. Formulated aminocarb was over 300 times more toxic than the technical grade material. Trichlorfon, aminocarb, and carbaryl were most toxic at a pH of 8.5. Water quality had no influence on the toxicity of acephate and fenitrothion. Water temperature had a minor influence on pesticide toxicity to the trout. Johnson and Julin⁹⁸ examined the influence of some experimental variables on the acute toxicity of toxaphene to fathead minnow (*Pimephales promelas*), bluegill, and channel catfish (*Ictalurus punctatus*). The LC50 values are given in Table 1. Water hardness and pH had little influence on these values. Temperature influenced the rate of response but not the degree of response. Early swim-up fry on the channel catfish was more sensitive than yolk-sac fry or fingerlings. The effect of body weight and water temperature on the acute toxicity of permethrin to rainbow trout (*Salmo gairdneri*) was compared by Kumaraguru and Beamish.⁹⁹ Using flow-through and measured concentrations the 96-hour LC50 values of 1-g trout exposed at 5°, 10°, 15°, and 20°C were 0.62, 0.69, 3.2, and 6.4 µg/l, respectively. The 96-hour LC50 values of 1-, 5-, 20-, 50-, and 200-g trout tested at 15°C were 3.2, 6.1, ~50, 287, and 314 µg/l, respectively. McLeese *et al.*¹⁰⁰ determined the static acute effects of permethrin, cypermethrin, and fenvalerate on Atlantic salmon, lobster (*Homarus americanus*), and shrimp (*C. sepiempinosa*). Fenvalerate was the most toxic and permethrin was the least toxic to the Atlantic salmon. The BCFs for Atlantic salmon for a 4-day exposure were 73 for permethrin, 7 for cypermethrin, and 200 for fenvalerate.

The acute toxicity of technical (95%) and commercial (50%) grade malathion to *Tilapia mossambica* was determined by

Table 1—A summary of the acute and chronic toxicity of inorganic and organic pollutants to freshwater fish.

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Acenaphthene ²⁶¹	Bluegill (<i>Lepomis macrochirus</i>)	96-h LC50 1.7 mg/l	S*	22	pH 6.7-7.8 HD ^b 32-48
Acephate (formulation, 75%) ⁹⁷	Cutthroat trout (<i>Salmo clarki</i>)	96-h LC50 >100 mg/l	S	12	pH 7.5 HD 40
Acephate (technical, 94%) ⁹⁷	Cutthroat trout	96-h LC50 >50 mg/l	S	12	pH 6.5 HD 40
		96-h LC50 >100 mg/l	S	7-12	pH 7.5 HD 40
		96-h LC50 >60 mg/l	S	12	pH 8.5 HD 40
		96-h LC50 >50 mg/l	S	12	pH 7.8 HD 320
Acetyl bromide ²⁶²	Fathead minnow (<i>Pimephales promelas</i>)	96-h LC50 40.6 mg/l	S	22	pH 7.2-7.9 HD 40-48
Acetyl chloride ²⁶²	Fathead minnow	96-h LC50 42.0 mg/l	S	22	pH 7.2-7.9 HD 40-48
Acrolein ²⁶¹	Bluegill	96-h LC50 0.09 mg/l	S	22	pH 6.7-7.8 HD 32-48
Acrylonitrile ²⁶¹	Bluegill	96-h LC50 10 mg/l	S	22	pH 6.7-7.8 HD 32-48
Adogen 283 ¹⁹⁷	Zebrafish (<i>Brachydanio rerio</i>)	96-h LC50 >5.6 mg/l	S	25	pH 7.3-8.5 HD 100
Adogen 383 ¹⁹⁷	Zebrafish	96-h LC50 >7.5 mg/l	S	25	pH 7.3-8.5 HD 100
Alamine 336 ¹⁹⁷	Zebrafish	96-h LC50 >3.2 mg/l	S	25	pH 7.3-8.5 HD 100
Aliquat 336 ¹⁹⁷	Zebrafish	96-h LC50 0.094-0.137 mg/l	S	25	pH 7.3-8.5 HD 100
Amberlite LA-1 ¹⁹⁷	Zebrafish	96-h LC50 >7.5	S	25	pH 7.3-8.5 HD 100
Aminocarb (formulation, 17%) ⁹⁷	Cutthroat trout	96-h LC50 0.09 mg/l	S	12	pH 7.5 HD 40
Aminocarb (technical, 98%) ⁹⁷	Cutthroat trout	96-h LC50 26 mg/l	S	12	pH 6.5 HD 40
		96-h LC50 27 mg/l	S	7	pH 7.5
		96-h LC50 28 mg/l	S	12	HD 40
		96-h LC50 1.3 mg/l	S	12	pH 8.5 HD 40
		96-h LC50 24 mg/l	S	12	pH 7.8 HD 320
Ammonium carbonate ²⁶²	Fathead minnow	96-h LC50 37 mg/l	S	22	pH 7.2-7.9 HD 40-48
Ammonium fluoride ²⁶²	Fathead minnow	96-h LC50 364 mg/l	S	22	pH 7.2-7.9 HD 40-48
Ammonium fluoborate ²⁶²	Fathead minnow	96-h LC50 >600 mg/l	S	22	pH 7.2-7.9 HD 40-48
Ammonium silicofluoride ²⁶²	Fathead minnow	96-h LC50 28.7 mg/l	S	22	pH 7.2-7.9 HD 40-48

(Table 1 Continued)

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Ammonium sulfate ²⁶²	Fathead minnow	96-h LC50 >600 mg/l	S	22	pH 7.2-7.9 HD 40-48
Aniline ²⁷²	Rainbow trout (<i>Salmo gairdneri</i>)	96-h LC50 20 mg/l	—	15	pH 7.4 HD 20
		96-h LC50 41 mg/l	—	15	pH 7.4 HD 320
Antimony trioxide ²⁶¹	Bluegill	96-h LC50 >530 mg/l	S	22	pH 6.7-7.8 HD 32-48
Antimony trioxide ²⁶²	Fathead minnow	96-h LC50 >833 mg/l	S	22	pH 7.2-7.9 HD 40-48
Antimycin ¹⁶²	Bighead carp (<i>Aristichyes nobilis</i>)	96-h LC50 0.6 µg/l	S	12	pH 7.5 HD 40
	Silver carp (<i>Hypophthalmichthys molitrix</i>)	96-h LC50 0.8 µg/l	S	12	
	Grass carp (<i>Ctenopharyngodon idella</i>)	96-h LC50 1.0 µg/l	S	12	
	Carp (<i>Cyprinus carpio</i>)	96-h LC50 0.6 µg/l	S	12	
Arocarb ¹⁰⁸	Carp	48-h LC50 >10 mg/l	—	—	—
	Goldfish (<i>Carassius auratus</i>)	48-h LC50 >10 mg/l	—	—	—
	Medaka (<i>Oryzias latipes</i>)	48-h LC50 >10 mg/l	—	—	—
	Pond loach (<i>Misgurnus anguillicaudatus</i>)	48-h LC50 >10 mg/l	—	—	—
Arsenic trisulfide ²⁶²	Fathead minnow	96-h LC50 135 mg/l	S	22	pH 7.2-7.9 HD 40-48
Benomyl ¹⁰⁶	Rainbow trout	72-h LC50 1.8 mg/l	—	—	—
	Carp	72-h LC50 190 mg/l	—	—	—
	Guppy (<i>Poecilia reticulata</i>)	72-h LC50 110 mg/l	—	—	—
Benthiocarb ¹⁰⁸	Carp	48-h LC50 1.5 mg/l	—	—	—
	Goldfish	48-h LC50 3.6 mg/l	—	—	—
	Medaka	48-h LC50 4.4 mg/l	—	—	—
	Pond loach	48-h LC50 7.2 mg/l	—	—	—
Benyl chloride ²⁶²	Fathead minnow	96-h LC50 4.7-7.8 mg/l	S	22	pH 7.2-7.9 HD 40-48
Benzoyl chloride ²³²	Fathead minnow	96-h LC50 34.1 mg/l	S	22	pH 7.2-7.9 HD 40-48
γBHC (lindane) ¹⁰⁸	Carp	48-h LC50 0.31 mg/l	—	—	—
	Goldfish	48-h LC50 0.12 mg/l	—	—	—
	Medaka	48-h LC50 0.12 mg/l	—	—	—
	Pond loach	48-h LC50 0.15 mg/l	—	—	—
γBHC (EC) (WP) (dust) (granule) ¹⁰⁸	Carp	48-h LC50 0.17 mg/l	—	—	—
		48-h LC50 0.95 mg/l	—	—	—
		48-h LC50 1.3 mg/l	—	—	—
		48-h LC50 1.7 mg/l	—	—	—
1BP (s-benzyl diisopropylphosphorothiolate) ¹⁰⁸	Carp	48-h LC50 >10 mg/l	—	—	—
	Goldfish	48-h LC50 12 mg/l	—	—	—
	Medaka	48-h LC50 7.2 mg/l	—	—	—
	Pond loach	48-h LC50 15 mg/l	—	—	—
Bromoform ²⁶¹	Bluegill	96-h LC50 29 mg/l	S	22	pH 6.7-7.8 HD 32-48
4-Bromophenyl phenyl ether ²⁶¹	Bluegill	96-h LC50 5.9 mg/l	S	22	pH 6.7-7.8 HD 32-48

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
di-n-Butylamine ²⁷²	Rainbow trout	96-h LC50 5.5 mg/l	—	15	pH 7.4 HD 20
		96-h LC50 37 mg/l	—	15	pH 7.4 HD 320
tert-Butylamine ²⁷²	Rainbow trout	96-h LC50 28 mg/l	—	15	pH 7.4 HD 20
		96-h LC50 270 mg/l	—	15	pH 7.4 HD 320
Butylbenzylphthalate ²⁶¹	Bluegill	96-h LC50 43 mg/l	S	22	pH 6.7–7.8 HD 32–48
Butyl butyrate ²⁶²	Fathead minnow	96-h LC50 11.6 mg/l	S	22	pH 7.2–7.9 HD 40–48
p-sec-Butylphenol ²⁷⁰	Atlantic Salmon (<i>Salmo salar</i>) Juvenile	96-h LC50 0.74 mg/l	S	10	—
di-n-Butylphthalate ²⁶¹	Bluegill	96-h LC50 1.2 mg/l	S	22	pH 6.7–7.8 HD 32–48
Butyraldehyde ²⁶²	Fathead minnow	96-h LC50 25.8 mg/l	S	22	pH 7.2–7.9 HD 40–48
Cadmium sulfate ¹⁸⁷	Zebrafish Juvenile	96-h LC50 1.7 mg/l	S	25	pH 7.3–8.5 HD 100
Captan ¹⁰⁸	Carp	48-h LC50 0.25 mg/l	—	—	—
	Goldfish	48-h LC50 0.04 mg/l	—	—	—
	Medaka	48-h LC50 1.0 mg/l	—	—	—
	Pond loach	48-h LC50 0.34 mg/l	—	—	—
Carbaryl ¹⁰⁸	Carp	48-h LC50 13 mg/l	—	—	—
	Goldfish	48-h LC50 >10 mg/l	—	—	—
	Medaka	48-h LC50 2.8 mg/l	—	—	—
	Pond loach	48-h LC50 13 mg/l	—	—	—
Carbaryl ¹⁰⁹	<i>Labeo rohita</i>				
	Larvae	96-h LC50 1 870 µg/l	S	26	pH 6.8–7.6
	Larvae	30-d MATC ^c 52–78 µg/l	S	26	HD 42
	<i>Cirrhina mrigala</i>				
	Larvae	96-h LC50 1 370 µg/l	S	26	
	Larvae	30-d MATC 52–78 µg/l	S	26	
	Carp				
	Larvae	96-h LC50 2 000 µg/l	S	26	
	Larvae	30-d MATC 52–78 µg/l	S	26	
	<i>Catla catla</i>				
	Larvae	96-h LC50 1 420 µg/l	S	26	
	Larvae	30-d MATC 52–78 µg/l	S	26	
Carbaryl (formulation, 49%) ⁹⁷	Cutthroat trout	96-h LC50 6.7 mg/l	S	12	pH 7.5 HD 40
Carbaryl (technical, 99%) ⁹⁷	Cutthroat trout	96-h LC50 5.0 mg/l	S	12	pH 6.5 HD 40
		96-h LC50 6.0 mg/l	S	7	pH 7.5
		96-h LC50 4.0 mg/l	S	12	HD 40
		96-h LC50 0.97 mg/l	S	12	pH 8.5 HD 40
		96-h LC50 4.0 mg/l	S	12	pH 7.8 HD 320

(Table 1 Continued)

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Carbofuran ¹⁰³	<i>Myxus vittatus</i>	96-h LC50 260 µg/l	S	18	pH 6.9-7.4
	<i>Ophiocephalus punctatus</i>	96-h LC50 180 µg/l	S	18	
Carbofuran ¹⁰⁸	Rainbow trout	48-h LC50 8.5 mg/l	—	—	—
	Carp	48-h LC50 11 mg/l	—	—	—
	Guppy	48-h LC50 3.4 mg/l	—	—	—
Carbon tetrachloride ²⁶¹	Bluegill	96-h LC50 27 mg/l	S	22	pH 6.7-7.8 HD 32-48
Cartap ¹⁰⁸	Carp	48-h LC50 0.78 mg/l	—	—	—
	Goldfish	48-h LC50 1.1 mg/l	—	—	—
	Medaka	48-h LC50 0.13 mg/l	—	—	—
	Pond loach	48-h LC50 0.11 mg/l	—	—	—
Chlordane ¹⁰⁸	<i>Labeo rohita</i>				
	Larvae	96-h LC50 20 µg/l	S	26	pH 6.8-7.6
	Larvae	30-d MATC 0.42-0.50 µg/l	S	26	
	<i>Cirrhina mrigala</i>				
	Larvae	96-h LC50 15 µg/l	S	26	
	Larvae	30-d MATC 0.42-0.50 µg/l	S	26	
	Carp				
	Larvae	96-h LC50 16 µg/l	S	26	
	Larvae	30-d MATC 0.42-0.50 µg/l	S	26	
	<i>Catla catla</i>				
Chlorine ²⁰⁷	Larvae	96-h LC50 12 µg/l	S	26	
	Larvae	30-d MATC 0.42-0.50 µg/l	S	26	
	Mosquitofish (<i>Gambusia affinis</i>)	1-h LC50 0.84 mg/l	F ^d	21	pH 7.4
Chlorobenzene ²⁶¹	Bluegill	96-h LC50 16 mg/l	S	22	pH 6.7-7.8 HD 32-48
bis(2-Chloroethyl)ether ²⁶¹	Bluegill	96-h LC50 600 mg/l	S	22	pH 6.7-7.8 HD 32-48
2-Chloroethyl vinyl ether ²⁶¹	Bluegill	96-h LC50 350 mg/l	S	22	pH 6.7-7.8 HD 32-48
4-Chloro-6-methyl-phenol ²⁶¹	Bluegill	96-h LC50 2.3 mg/l	S	22	pH 6.7-7.8 HD 32-48
1-Chloronaphthalene ²⁶¹	Bluegill	96-h LC50 2.3 mg/l	S	22	pH 6.7-7.8 HD 32-48
2-Chlorophenol ²⁶¹	Bluegill	96-h LC50 6.6 mg/l	S	22	pH 6.7-7.8 HD 32-48
2-Chlorophenol ²⁶⁹	Fathead minnow	48-h LC50 9.7 mg/l 96-h LC50 11-13 mg/l 192-h LC50 6.3 mg/l	S F F	22-25 25 25	HD 43-49
4-Chlorophenol ²⁶¹	Bluegill	96-h LC50 3.8 mg/l	S	22	pH 6.7-7.8 HD 32-48
Chromium ¹⁸⁵	Zebrafish	96-h LC50 58.5 mg/l	S	25	pH 8.0 HD 350
Cobaltous bromide ²⁶²	Fathead minnow	96-h LC50 91.9 mg/l	S	22	pH 7.2-7.9 HD 40-48
Cobaltous formate ²⁶²	Fathead minnow	96-h LC50 32.2 mg/l	S	22	pH 7.2-7.9 HD 40-48
Copper ¹⁸⁶	Guppy	96-h LC50 1.23 mg/l	S	17	pH 7.3 HD 160

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Copper sulfate ¹⁹⁴	<i>Rasbora daniconius</i> <i>nelgeriensis</i>	96-h LC50 0.20 mg/l	S	25	pH 7.5 HD 240
Cupric acetate ²⁶²	Fathead minnow	96-h LC50 0.39 mg/l	S	22	pH 7.2–7.9 HD 40–48
Cyclohexylamine ²⁷²	Rainbow trout	96-h LC50 44 mg/l	—	15	pH 7.4 HD 20
		96-h LC50 90 mg/l	—	15	pH 7.4 HD 320
2,4-D ¹⁵²	Carp	96-h LC50 5.1–35 mg/l	R ^a	39–17	pH 7.8–8.4
2,4-D ¹⁵¹	<i>Labeo boga</i>	96-h LC50 3.8 mg/l	R	28	pH 8.0
	<i>Rasbora nelgeriensis</i>	96-h LC50 5.6 mg/l	R	28	
	Carp	96-h LC50 20 mg/l	R	28	
2,4-D (sodium salt) ¹⁰⁸	Carp	48-h LC50 >40 mg/l	—	—	—
	Goldfish	48-h LC50 >40 mg/l	—	—	—
	Medaka	48-h LC50 >40 mg/l	—	—	—
	Pond loach	48-h LC50 >40 mg/l	—	—	—
DDT ¹⁰⁸	Carp	48-h LC50 0.25 mg/l	—	—	—
	Goldfish	48-h LC50 0.07 mg/l	—	—	—
	Medaka	48-h LC50 0.01 mg/l	—	—	—
	Pond loach	48-h LC50 0.24 mg/l	—	—	—
DDT (EC)	Carp	48-h LC50 0.35 mg/l	—	—	—
(WP)		48-h LC50 2.0 mg/l	—	—	—
(dust) ¹⁰⁸		48-h LC50 5.0 mg/l	—	—	—
Diazinon ¹⁰⁸	Carp	48-h LC50 3.2 mg/l	—	—	—
	Goldfish	48-h LC50 5.1 mg/l	—	—	—
	Medaka	48-h LC50 5.3 mg/l	—	—	—
	Pond loach	48-h LC50 0.50 mg/l	—	—	—
Diazinon ¹¹⁸	Rainbow trout	14-d BCF ¹ 63	F	22	pH 7.4
	Carp	14-d BCF 120	F	22	
	Loach	14-d BCF 26	F	25	
3,4-Dichloroaniline ²⁷¹	Guppy Juvenile	48-h LC50 9.5 mg/l	—	—	pH 8.0
		96-h LC50 8.7–9.0 mg/l	—	—	
		7-d LC50 8.2–8.5 mg/l	—	—	
		4-wk LC50 5.5 mg/l	—	—	
		8-wk LC50 3.2 mg/l	—	—	
		4-mo LC50 2.1 mg/l	—	—	
		4-mo EC50 0.55 mg/l	—	—	
1,2-Dichlorobenzene ²⁶¹	Bluegill	96-h LC50 5.6 mg/l	S	22	pH 6.7–7.8 HD 32–48
1,3-Dichlorobenzene ²⁶¹	Bluegill	96-h LC50 5.0 mg/l	S	22	pH 6.7–7.8 HD 32–48
1,4-Dichlorobenzene ²⁶¹	Bluegill	96-h LC50 4.3 mg/l	S	22	pH 6.7–7.8 HD 32–48
o-Dichlorobenzene ²⁶²	Fathead minnow	96-h LC50 57 mg/l	S	22	pH 7.2–7.9 HD 40–48
p-Dichlorobenzene ²⁶²	Fathead minnow	96-h LC50 30 mg/l	S	22	pH 7.2–7.9 HD 40–48
1,2-Dichloroethane ²⁶¹	Bluegill	96-h LC50 430 mg/l	S	22	pH 6.7–7.8 HD 32–48

(Table 1 Continued)

Table 1—(Continued)

Pollutant	Species	Results		Exposure type	Temp. (°C)	Test conditions
1,1-Dichloroethylene ²⁶¹	Bluegill	96-h LC50	74 mg/l	S	22	pH 6.7-7.8 HD 32-48
1,1-Dichloroethylene ²⁶⁴	Fathead minnow	96-h LC50	169 mg/l	S	12	pH 7.9
		96-h LC50	108 mg/l	F	—	HD 100
		13-d Threshold	29 mg/l	F	—	
1,2-Dichloroethylene ²⁶¹	Bluegill	96-h LC50	140 mg/l	S	22	pH 6.7-7.8 HD 32-48
Dichloromethane ²⁶¹	Bluegill	96-h LC50	220 mg/l	S	22	pH 6.7-7.8 HD 32-48
2,4-Dichloro-6-methyl ²⁶¹	Bluegill	96-h LC50	1.6 mg/l	S	22	pH 6.7-7.8 HD 32-48
2,4-Dichlorophenol ²⁶¹	Bluegill	96-h LC50	2.0 mg/l	S	22	pH 6.7-7.8 HD 32-48
2,4-Dichlorophenol ²⁶⁹	Fathead minnow	48-h LC50	8.4 mg/l	S	22-25	HD 43-49
		96-h LC50	8.2-8.3 mg/l	F	25	
		192-h LC50	6.5 mg/l	F	25	
1,1-Dichloropropane ²⁶¹	Bluegill	96-h LC50	98 mg/l	S	22	pH 6.7-7.8 HD 32-48
1,2-Dichloropropane ²⁶¹	Bluegill	96-h LC50	280 mg/l	S	22	pH 6.7-7.8 HD 32-48
1,3-Dichloropropane ²⁶¹	Bluegill	96-h LC50	>520 mg/l mg/l	S	22	pH 6.7-7.8 HD 32-48
1,3-Dichloropropene ²⁶¹	Bluegill	96-h LC50	6.1 mg/l	S	22	pH 6.7-7.8 HD 32-48
Dichlorvos ¹⁰³	<i>Mystus vittatus</i>	96-h LC50	400 µg/l	S	18	pH 6.9-7.4
	<i>Ophiocephalus punctatus</i>	96-h LC50	2 040 µg/l	S	18	
Dieldrin ²⁷¹	Guppy Juvenile	48-h LC50	0.005-0.012 mg/l	—	—	pH 8.0
		96-h LC50	0.003-0.007 mg/l	—	—	
		7-d LC50	0.003-0.005 mg/l	—	—	
		14-d LC50	0.003 mg/l	—	—	
		2-mo LC50	0.003 mg/l	—	—	
		48-h LC50	0.035-0.073 mg/l	—	—	
	Adult	7-d LC50	0.009-0.030 mg/l	—	—	
Diethylamine ²⁷²	Rainbow trout	96-h LC50	25 mg/l	—	15	pH 7.4 HD 20
		96-h LC50	182 mg/l	—	15	pH 7.4 HD 320
Diethylphthalate ²⁶¹	Bluegill	96-h LC50	110 mg/l	S	22	pH 6.7-7.8 HD 32-48
Diisopropylamine ²⁷²	Rainbow trout	96-h LC50	37 mg/l	—	15	pH 7.4 HD 20
		96-h LC50	196 mg/l	—	15	pH 7.4 HD 320
Dimethylamine ²⁷²	Rainbow trout	96-h LC50	17 mg/l	—	15	pH 7.4 HD 20
		96-h LC50	118 mg/l	—	15	pH 7.4 HD 320
2,4-Dimethylphenol ²⁶¹	Bluegill	96-h LC50	7.8 mg/l	S	22	pH 6.7-7.8 HD 32-48

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
2,4-Dimethylphenol ²⁶⁰	Fathead minnow	48-h LC50	9.5 mg/l	S	21-25 pH 7.6-9.0
		96-h LC50	17 mg/l	F	25 HD 43-49
		192-h LC50	13-14 mg/l	F	25
2,6-Dimethylphenol ²⁶⁰	Fathead minnow	48-h LC50	6.7 mg/l	S	21-25 pH 7.3-8.6
		96-h LC50	>27 mg/l	F	25 HD 43-49
		192-h LC50	21-23 mg/l	F	25
Dimethylphthalate ²⁶¹	Bluegill	96-h LC50	50 mg/l	S	22 pH 6.7-7.8 HD 32-48
m-Dinitrobenzene ²⁶²	Fathead minnow	96-h LC50	12.7 mg/l	S	22 pH 7.2-7.9 HD 40-48
2,4-Dinitro-6-methylphenol ²⁶¹	Bluegill	96-h LC50	0.23 mg/l	S	22 pH 6.7-7.8 HD 32-48
4,6-Dinitro-o-cresol ²⁶⁰	Fathead minnow	48-h LC50	8.6 mg/l	S	22-25 HD 43-49
		96-h LC50	1.9-2.2 mg/l	F	25
		192-h LC50	1.3-1.7 mg/l	F	25
Dinitrophenol ²⁶⁷	Patra (<i>Notopterus notopterus</i>)	96-h LC50	1.34 mg/l	S	—
Dinitrophenol ²⁶⁸	Khanda (<i>Colisa fasciatus</i>)	96-h LC50	8.60 mg/l	S	24 pH 6.8-7.6
	Singli (<i>Saccobranhus fossilis</i>)	96-h LC50	6.85 mg/l	S	24 HD 66
	Patra	96-h LC50	1.34 mg/l	S	24
Dinitrophenol (technical) ²⁶⁰	Khanda	96-h LC50	1.8 mg/l	R	— pH 4.6
	Singli	96-h LC50	1.2 mg/l	R	—
	Patra	96-h LC50	0.06 mg/l	R	—
	Khanda	96-h LC50	7.5 mg/l	R	— pH 6.0
	Singli	96-h LC50	5.9 mg/l	R	—
	Patra	96-h LC50	0.9 mg/l	R	—
	Khanda	96-h LC50	8.6 mg/l	R	— pH 7.3
	Singli	96-h LC50	6.9 mg/l	R	—
	Patra	96-h LC50	1.3 mg/l	R	—
	Khanda	96-h LC50	12.1 mg/l	R	— pH 8.8
	Singli	96-h LC50	8.6 mg/l	R	—
	Patra	96-h LC50	2.9 mg/l	R	—
2,4-Dinitrophenol ²⁶¹	Bluegill	96-h LC50	0.62 mg/l	S	22 pH 6.7-7.8 HD 32-48
2,4-Dinitrophenol ²⁶⁰	Fathead minnow	48-h LC50	7.3 mg/l	S	22-25 HD 43-49
		96-h LC50	17 mg/l	F	25
		192-h LC50	16 mg/l	F	25
2,3-Dinitrotoluene ²⁶¹	Bluegill	96-h LC50	0.33 mg/l	S	22 pH 6.7-7.8 HD 32-48
Dinoseb ¹⁰⁸	Carp	48-h LC50	0.07 mg/l	—	—
	Goldfish	48-h LC50	0.07 mg/l	—	—
	Medaka	48-h LC50	0.15 mg/l	—	—
	Pond loach	48-h LC50	0.18 mg/l	—	—
Dioxacarb ¹⁰⁶	Rainbow trout	48-h LC50	2.7 mg/l	—	—
	Carp	48-h LC50	25.5 mg/l	—	—
	Guppy	48-h LC50	36 mg/l	—	—
1,2-Diphenylhydrazine ²⁶¹	Bluegill	96-h LC50	0.27 mg/l	S	22 pH 6.7-7.8 HD 32-48
p-Dodecylphenol ²⁷⁰	Atlantic salmon Juvenile	96-h LC50	0.14 mg/l	S	10

(Table 1 Continued)

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
EDTA ²⁶²	Fathead minnow	96-h LC50 59.8 mg/l	S	22	pH 7.2-7.9 HD 40-48
Endosulfan ¹⁰⁸	Carp	48-h LC50 0.007 mg/l	—	—	—
	Goldfish	48-h LC50 0.000 1 mg/l	—	—	—
	Medaka	48-h LC50 0.009 mg/l	—	—	—
	Pond loach	48-h LC50 0.001 mg/l	—	—	—
Endosulfan ¹⁰²	Catfish (<i>Clarias batrachus</i>)	96-h LC50 14 µg/l	S	20	pH 6.9-7.2 HD 118-122
Endosulfan ¹²⁴	<i>Macrogathus aculeatus</i>	96-h LC50 3.5 µg/l	F	28	pH 8.4 HD 152
Endosulfan ¹⁰⁰	<i>Mystus vittatus</i>	96-h LC50 0.79 µg/l	S	28	pH 6.9-7.4
	<i>Ophiocephalus punctatus</i>	96-h LC50 21 µg/l	S	18	
Endosulfan (technical) ¹⁰⁴	Sauli (<i>Channa punctatus</i>)	96-h LC50 4.8 µg/l	F	30	pH 8.4 HD 152
Endosulfan (35% EC) ¹⁰⁴	Sauli	96-h LC50 2.5 µg/l	F	30	pH 8.4 HD 152
Endosulfan (4% dust) ¹⁰⁴	Sauli	96-h LC50 16 µg/l	F	30	pH 8.4 HD 152
Endosulfan (isomer A) ¹⁰⁴	Sauli	96-h LC50 0.16 µg/l	F	30	pH 8.4 HD 152
Endosulfan (isomer B) ¹⁰⁴	Sauli	96-h LC50 6.6 µg/l	F	30	pH 8.4 HD 152
Endrin ¹⁰⁸	Carp	48-h LC50 0.000 8 mg/l	—	—	—
	Goldfish	48-h LC50 0.001 mg/l	—	—	—
	Medaka	48-h LC50 0.001 4 mg/l	—	—	—
	Pond loach	48-h LC50 0.004 9 mg/l	—	—	—
Ethylbenzene ²⁶¹	Bluegill	96-h LC50 150 mg/l	S	22	pH 6.7-7.8 HD 32-48
Ethylenediamine ²⁶²	Fathead minnow	96-h LC50 115.7 mg/l	S	22	pH 7.2-7.9 HD 40-48
bis(2-Ethylhexyl)phthalate ²⁶¹	Bluegill	96-h LC50 >770 mg/l	S	22	pH 6.7-7.8 HD 32-48
Fenitrothion ¹⁰⁸	Carp	48-h LC50 8.2 mg/l	—	—	—
	Goldfish	48-h LC50 3.4 mg/l	—	—	—
	Medaka	48-h LC50 7.0 mg/l	—	—	—
	Pond loach	48-h LC50 4.8 mg/l	—	—	—
Fenitrothion (EC) (WP) (dust) ¹⁰⁸	Carp	48-h LC50 2.8 mg/l	—	—	—
	Carp	48-h LC50 5.0 mg/l	—	—	—
	Carp	48-h LC50 4.3 mg/l	—	—	—
Fenitrothion (formulation, 87%) ⁹⁷	Cutthroat trout	96-h LC50 2.9 mg/l	S	12	pH 7.5 HD 40
Fenitrothion (technical, 95%) ⁹⁷	Cutthroat trout	96-h LC50 2.8 mg/l	S	12	pH 6.5 HD 40
		96-h LC50 2.7 mg/l	S	7	pH 7.5
		96-h LC50 2.9 mg/l	S	12	HD 40
		96-h LC50 2.6 mg/l	S	12	pH 8.5 HD 40
		96-h LC50 2.8 mg/l	S	12	pH 7.8 HD 320

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Fentin acetate ¹⁰⁸	Carp	48-h LC50 0.064 mg/l	—	—	—
	Goldfish	48-h LC50 0.062 mg/l	—	—	—
	Medaka	48-h LC50 0.097 mg/l	—	—	—
	Pond loach	48-h LC50 0.290 mg/l	—	—	—
Ferbam ¹⁰⁸	Carp	48-h LC50 0.09 mg/l	—	—	—
	Goldfish	48-h LC50 0.13 mg/l	—	—	—
	Medaka	48-h LC50 0.05 mg/l	—	—	—
	Pond loach	48-h LC50 1.8 mg/l	—	—	—
Fluoranthene ²⁶¹	Bluegill	96-h LC50 4.0 mg/l	S	22	pH 6.7–7.8 HD 32–48
Fluoroacetamide ¹⁰⁸	Carp	48-h LC50 >40 mg/l	—	—	—
	Goldfish	48-h LC50 >40 mg/l	—	—	—
	Medaka	48-h LC50 >40 mg/l	—	—	—
	Pond loach	48-h LC50 >40 mg/l	—	—	—
GD-174 (2-(digeranyl-amino)-ethanol) ¹⁶²	Bighead carp	96-h LC50 0.36 mg/l	S	12	pH 7.5
	Silver carp	96-h LC50 0.55 mg/l	S	12	HD 40
	Grass carp	96-h LC50 0.21 mg/l	S	12	
	Carp	96-h LC50 0.05 mg/l	S	12	
HDEHP ¹⁸⁷	Zebrafish	96-h LC50 >56 mg/l	S	25	pH 7.3–8.5 HD 100
Hexachloroethane ²⁶¹	Bluegill	96-h LC50 0.98 mg/l	S	22	pH 6.7–7.8 HD 32–48
p-Hexylphenol ²⁷⁰	Atlantic salmon Juvenile	96-h LC50 0.19 mg/l	S	10	—
Hydrogen cyanide ⁵⁵	Rainbow trout	18-d Decreased growth 0.01 mg/l	R	13	pH 7.9 HD 128
Hydrothol-191 (mono-n,n-di-methylakylamine salt of endothal) ¹⁵⁴	Golden shiner (<i>Notemigonus crysoleucas</i>)	120-h LC50 1.6 mg/l	F	—	pH 7.2 HD 20
		120-h LC50 0.32 mg/l	F	—	pH 8.2 HD 275
Idet 5L ²⁷³	Saudi	96-h LC50 22.7–35.0 mg/l	S	—	pH 6.9–7.2
Iron methanoarsenate ¹⁰⁸	Carp	48-h LC50 >40 mg/l	—	—	—
	Goldfish	48-h LC50 >40 mg/l	—	—	—
	Medaka	48-h LC50 >40 mg/l	—	—	—
	Pond loach	48-h LC50 >40 mg/l	—	—	—
Isophorone ²⁶¹	Bluegill	96-h LC50 220 mg/l	S	22	pH 6.7–7.8 HD 32–48
Kasugamycin ¹⁰⁸	Carp	48-h LC50 >40 mg/l	—	—	—
	Goldfish	48-h LC50 >40 mg/l	—	—	—
	Medaka	48-h LC50 >40 mg/l	—	—	—
	Pond loach	48-h LC50 3.5 mg/l	—	—	—
Kepone ¹¹⁰	Fathead minnow				
	Larvae	96-h LC50 420 µg/l	S	25	pH 7.4
	Adult	96-h LC50 340 µg/l	F	25	HD 272
	Larvae	96-h EC50 18 µg/l	F	25	
	Adult	96-h EC50 41 µg/l	F	25	
	Larvae	16-20-d LC50 20 µg/l	F	25	
	Adult	26-30-d LC50 31 µg/l	F	25	
		MATC 1.2–3.1 µg/l	F	25	
		60-d BCF 1 100–2 200	F	25	

(Table 1 Continued)

Water Pollution

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Komeen (copper-ethylenediamine complex) ¹⁵⁴	Golden shiner	96-h LC50 67 mg/l	F	—	pH 7.2 HD 20
		96-h LC50 630 mg/l	F	—	pH 8.2 HD 275
Lead acetate ²⁶²	Fathead minnow	96-h LC50 43.6 mg/l	S	22	pH 7.2-7.9 HD 40-48
Lead fluoborate ²⁶²	Fathead minnow	96-h LC50 18 mg/l	S	22	pH 7.2-7.9 HD 40-48
Lead nitrate ²⁵⁸	<i>Ophiocephalus punctatus</i>	96-h LC50 13.2 mg/l	S	20	pH 7.1
	<i>Heteropneustes fossilis</i>	96-h LC50 12 mg/l	S	20	HD 160
Melathion (technical, 95%) ¹⁰¹	<i>Tilapia mossambica</i>	48-h LC50 5.6 mg/l	S	26-28	pH 7.0 HD 140
Melathion (commercial, 50%) ¹⁰¹	<i>Tilapia mossambica</i>	48-h LC50 0.38 mg/l	S	26-28	pH 7.0 HD 140
Mancozeb (zinc manganate ethylene-bis-dithiocarbamidan) ¹⁰⁶	Rainbow trout	48-h LC50 1.9 mg/l	—	—	—
	Carp	48-h LC50 24 mg/l	—	—	—
	Guppy	48-h LC50 2.2 mg/l	—	—	—
Manganese sulfate ²⁴²	Kharia Adult	96-h LC50 2 850 mg/l	S	25	pH 7.3 HD 120
Mercuric acetate ²⁶²	Fathead minnow	96-h LC50 0.19 mg/l	S	22	pH 7.2-7.9 HD 40-48
Mercuric chloride ²¹¹	Goldfish	24-h LC50 0.4 mg/l	S	—	—
Mercuric thiocyanate ²⁶²	Fathead minnow	96-h LC50 0.15 mg/l	S	22	pH 7.2-7.9 HD 40-48
Methomyl ¹⁰⁶	Carp	48-h LC50 2.8 mg/l	—	—	—
	Goldfish	48-h LC50 2.7 mg/l	—	—	—
	Medaka	48-h LC50 0.87 mg/l	—	—	—
	Pond loach	48-h LC50 1.5 mg/l	—	—	—
3-Methoxyphenol ²⁶⁰	Fathead minnow	48-h LC50 8.7 mg/l	S	22-25	pH 7.5-8.6
		96-h LC50 75-77 mg/l	F	25	HD 43-49
		192-h LC50 55-60 mg/l	F	25	
Methyl demeton ¹⁰⁶	<i>Labeo rohita</i>				
	Larvae	96-h LC50 1 560 µg/l	S	26	pH 6.8-7.6
	Larvae	30-d MATC 40-50 µg/l	S	26	HD 42
	<i>Cirrhina mrigala</i>				
	Larvae	96-h LC50 1 240 µg/l	S	26	
	Larvae	30-d MATC 30-40 µg/l	S	26	
	Carp				
	Larvae	96-h LC50 1 420 µg/l	S	26	
	Larvae	30-d MATC 40-50 µg/l	S	26	
	<i>Catla catla</i>				
	Larvae	96-h LC50 1 600 µg/l	S	26	
	Larvae	30-d MATC 40-50 µg/l	S	26	
Methylmercuric chloride ²¹¹	Goldfish	24-h LC50 0.08 mg/l	S	—	—
Methyl parathion ¹⁰⁶	Carp	48-h LC50 12 mg/l	S	28-30	pH 7.1-7.3
Methyl parathion ¹²⁸	<i>Heteropneustes fossilis</i>	96-h LC50 7.0 mg/l	S	24	pH 7.7
					HD 117
Mirex ¹¹⁰	Fathead minnow	120-d BCF 12 000-28 000	F	25	pH 7.4 HD 272

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Morpholine ²⁷²	Rainbow trout	96-h LC50 180 mg/l	—	15	pH 7.4 HD 20
		96-h LC50 380 mg/l	—	15	pH 7.4 HD 320
Nickel ¹⁸⁰	<i>Puntius sophore</i>	96-h LC50 13.6 mg/l	S	21	pH 7.4
	<i>Rasbora daniconius</i>	96-h LC50 48.8 mg/l	S	21	HD 260
	<i>natogerensis</i>	96-h LC50 24.5 mg/l	S	21	
	Guppy	96-h LC50 34.9 mg/l	S	21	
p-Nitroaniline ²⁶²	Fathead minnow	96-h LC50 106 mg/l	S	22	pH 7.2-7.9 HD 40-48
o-Nitroanisole ²⁶²	Fathead minnow	96-h LC50 216 mg/l	S	22	pH 7.2-7.9 HD 40-48
Nitrobenzaldehyde ²⁶²	Fathead minnow	96-h LC50 5.8 mg/l	S	22	pH 7.2-7.9 HD 40-48
Nitrobenzene ²⁶¹	Bluegill	96-h LC50 43 mg/l	S	22	pH 6.7-7.8 HD 32-48
Nitrofen ¹⁰⁸	Carp	48-h LC50 2.1 mg/l	—	—	—
	Goldfish	48-h LC50 1.4 mg/l	—	—	—
	Medaka	48-h LC50 2.5 mg/l	—	—	—
	Pond loach	48-h LC50 5.5 mg/l	—	—	—
Nitromethane ²⁶²	Fathead minnow	96-h LC50 >650 mg/l	S	22	pH 7.2-7.9 HD 40-48
1-Nitronaphthalene ²⁶²	Fathead minnow	96-h LC50 9.0 mg/l	S	22	pH 7.2-7.9 HD 40-48
4-Nitrophenol ²⁶¹	Bluegill	96-h LC50 8.3 mg/l	S	22	pH 6.7-7.8 HD 32-48
4-Nitrophenol ²⁶⁰	Fathead minnow	48-h LC50 10 mg/l	S	22-25	HD 43-49
		96-h LC50 59-62 mg/l	F	25	
		192-h LC50 49-50 mg/l	F	25	
2-Nitropropane ²⁶²	Fathead minnow	96-h LC50 >612 mg/l	S	22	pH 7.2-7.9 HD 40-48
n-Nitrosodiphenylamine ²⁶¹	Bluegill	96-h LC50 5.8 mg/l	S	22	pH 6.7-7.8 HD 32-48
p-Nonylphenol ²⁷⁰	Atlantic salmon Juvenile	96-h LC50 0.19 mg/l	S	10	—
		96-h LC50 0.13-0.16 mg/l	F	10	—
Noxifish ¹⁶²	Bighead carp	96-h LC50 44 µg/l	S	12	pH 7.5
	Silver carp	96-h LC50 56 µg/l	S	12	HD 40
	Grass carp	96-h LC50 85 µg/l	S	12	
	Carp	96-h LC50 50 µg/l	S	12	
Octachloronaphthalene ²⁶¹	Bluegill	96-h LC50 >600 mg/l	S	22	pH 6.7-7.8 HD 32-48
Paraquat ¹⁰⁸	Carp	48-h LC50 >40 mg/l	—	—	—
	Goldfish	48-h LC50 >10 mg/l	—	—	—
	Medaka	48-h LC50 7.0 mg/l	—	—	—
	Pond loach	48-h LC50 35 mg/l	—	—	—

(Table 1 Continued)

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Parathion ¹⁰⁸	Carp	48-h LC50 4.5 mg/l	—	—	—
	Goldfish	48-h LC50 1.7 mg/l	—	—	—
	Medaka	48-h LC50 2.9 mg/l	—	—	—
	Pond loach	48-h LC50 1.4 mg/l	—	—	—
Pentachlorobenzene ²⁶¹	Bluegill	96-h LC50 0.25 mg/l	S	22	pH 6.7–7.8 HD 32–48
Pentachloroethane ²⁶¹	Bluegill	96-h LC50 7.2 mg/l	S	22	pH 6.7–7.8 HD 32–48
Pentachlorophenol ²⁷¹	Guppy Juvenile	48-h LC50 1.05 mg/l	—	—	pH 8.0
		96-h LC50 0.72–0.88 mg/l	—	—	—
		7-d LC50 0.58–0.80 mg/l	—	—	—
		60-d No effect on growth <0.18 mg/l	—	—	—
	Adult	48-h LC50 0.82 mg/l	—	—	—
		96-h LC50 0.45 mg/l	—	—	—
		7-d LC50 0.36 mg/l	—	—	—
		—	—	—	—
Pentachlorophenol ²⁶⁹	Fathead minnow	48-h LC50 7.9 mg/l	S	22–25	pH 7.4–8.33
		96-h LC50 0.22–0.23 mg/l	F	25	HD 43–49
		192-h LC50 0.20–0.22 mg/l	F	25	—
Pentachlorophenol ²⁶⁷	Patra	96-h LC50 0.083 mg/l	S	—	—
Pentachlorophenol ²⁶⁶	Kharda	96-h LC50 0.45 mg/l	S	24	pH 6.8–7.6
	Singli	96-h LC50 0.29 mg/l	S	24	HD 66
	Patra	96-h LC50 0.083 mg/l	S	24	—
Pentachlorophenol (technical) ²⁶⁸	Kharda	96-h LC50 0.092 mg/l	R	—	pH 4.6
	Singli	96-h LC50 0.06 mg/l	R	—	—
	Patra	96-h LC50 0.004 mg/l	R	—	—
	Kharda	96-h LC50 0.034 mg/l	R	—	pH 6.0
	Singli	96-h LC50 0.18 mg/l	R	—	—
	Patra	96-h LC50 0.05 mg/l	R	—	—
	Kharda	96-h LC50 0.45 mg/l	R	—	pH 7.3
	Singli	96-h LC50 0.29 mg/l	R	—	—
	Patra	96-h LC50 0.08 mg/l	R	—	—
	Kharda	96-h LC50 0.85 mg/l	R	—	pH 8.8
	Singli	96-h LC50 0.75 mg/l	R	—	—
	Patra	96-h LC50 0.37 mg/l	R	—	—
Permethrin ⁹⁰	Rainbow trout	—	—	—	—
	1 g	96-h LC50 0.62–6.43 µg/l	F	15–20	pH 8.2
	200 g	96-h LC50 314 µg/l	F	15	HD 358–363
Permethrin ¹¹⁶	Rainbow trout	24-h LD50 14 mg/kg	IP ⁹⁰	12	—
		24-h LD50 18 µg/l	S	12	—
Permethrin (cis-isomer) ¹¹⁸	Rainbow trout	24-h LD50 22 mg/kg	IP	12	—
		24-h LD50 25 µg/l	S	12	—
Permethrin (trans-isomer) ¹¹⁸	Rainbow trout	24-h LD50 7 mg/kg	IP	12	—
		24-h LD50 14 µg/l	S	12	—
Phenol ²⁶⁹	Fathead minnow	48-h LC50 8.3 mg/l	S	22–25	HD 43–49
		96-h LC50 28–29 mg/l	F	25	—
		192-h LC50 22–23 mg/l	F	25	—
Phenol ²⁶⁶	Kharda	96-h LC50 32.7 mg/l	S	24	pH 6.8–7.6
	Singli	96-h LC50 39.4 mg/l	S	24	HD 66
	Patra	96-h LC50 12.5 mg/l	S	24	—
Phenol ²⁶⁷	Patra	96-h LC50 12.5 mg/l	S	—	—

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Phenol ²⁶⁸	Khorda	96-h LC50 9.1 mg/l	R	—	pH 4.6
	Singli	96-h LC50 13 mg/l	R	—	
	Patra	96-h LC50 2.5 mg/l	R	—	
	Khorda	96-h LC50 28.9 mg/l	R	—	pH 6.0
	Singli	96-h LC50 37.4 mg/l	R	—	
	Patra	96-h LC50 11.5 mg/l	R	—	
	Khorda	96-h LC50 32.7 mg/l	R	—	pH 7.3
	Singli	96-h LC50 39.4 mg/l	R	—	
	Patra	96-h LC50 12.5 mg/l	R	—	
	Khorda	96-h LC50 49.2 mg/l	R	—	pH 8.8
	Singli	96-h LC50 54.1 mg/l	R	—	
	Patra	96-h LC50 29.2 mg/l	R	—	
Phenyl mercury acetate ¹⁰⁸	Carp	48-h LC50 0.08 mg/l	—	—	—
	Goldfish	48-h LC50 0.07 mg/l	—	—	—
	Medaka	48-h LC50 0.13 mg/l	—	—	—
	Pond loach	48-h LC50 0.50 mg/l	—	—	—
Primene JM-T ¹⁹⁷	Zebrafish	96-h LC50 0.47 mg/l	S	25	pH 7.3–8.5 HD 100
Propanil ¹⁰⁸	Carp	48-h LC50 13 mg/l	—	—	—
	Goldfish	48-h LC50 14 mg/l	—	—	—
	Medaka	48-h LC50 11 mg/l	—	—	—
	Pond loach	48-h LC50 8.3 mg/l	—	—	—
Resorcinol ²⁶²	Fathead minnow	96-h LC50 60 mg/l	S	22	pH 7.2–7.9 HD 40–48
Rotenone ¹⁰⁸	Carp	48-h LC50 0.032 mg/l	—	—	—
	Goldfish	48-h LC50 0.033 mg/l	—	—	—
	Medaka	48-h LC50 0.030 mg/l	—	—	—
	Pond loach	48-h LC50 0.037 mg/l	—	—	—
Salicylanilide ¹⁶²	Bighead carp	96-h LC50 4.7 µg/l	S	12	pH 7.5
	Silver carp	96-h LC50 4.4 µg/l	S	12	HD 40
	Grass carp	96-h LC50 9.4 µg/l	S	12	
	Carp	96-h LC50 1.5 µg/l	S	12	
Selenomethionine ²¹¹	Goldfish	24-h LC50 >2.6 mg/l	S	—	—
Silver nitrate ²⁶¹	Bluegill	96-h LC50 0.06 mg/l	S	22	pH 6.7–7.8 HD 32–48
Simazine ¹⁰⁸	Carp	48-h LC50 >40 mg/l	—	—	—
	Goldfish	48-h LC50 >40 mg/l	—	—	—
	Medaka	48-h LC50 >40 mg/l	—	—	—
	Pond loach	48-h LC50 >40 mg/l	—	—	—
Sodium arsenate ²³²	Mosquitofish	96-h LC50 49 mg/l	—	—	—
Sodium hypochlorite (4–6 % solution) ²⁶²	Fathead minnow	96-h LC50 5.9 mg/l	S	22	pH 7.2–7.9 HD 40–48
Sodium pentachlorophenol ¹⁰⁸	Carp	48-h LC50 0.12 mg/l	—	—	—
	Goldfish	48-h LC50 0.12 mg/l	—	—	—
	Medaka	48-h LC50 0.08 mg/l	—	—	—
	Pond loach	48-h LC50 0.12 mg/l	—	—	—
Sodium selenite ²¹¹	Goldfish	24-h LC50 41 mg/l	S	—	—
Sulfamic acid ²⁶²	Fathead minnow	96-h LC50 70.3 mg/l	S	22	pH 7.2–7.9 HD 40–48

(Table 1 Continued)

Water Pollution

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Sulfanilic acid ²⁶²	Fathead minnow	96-h LC50 100.4 mg/l	S	22	pH 7.2–7.9 HD 40–48
Swanic 6L ²⁷³	Saudi	96-h LC50 15 mg/l	S	—	pH 6.9–7.2
	Singii	96-h LC50 6.0–6.4 mg/l	S	—	
Swaryl 10C ²⁷³	Saudi	96-h LC50 19.6–19.7 mg/l	S	—	pH 6.9–7.2
	Singii	96-h LC50 11.2 mg/l	S	—	
Swascofix CD-38 ²⁷³	Saudi	96-h LC50 26.9–27.0 mg/l	S	—	pH 6.9–7.2
	Singii	96-h LC50 17.4–17.7 mg/l	S	—	
Swascol 1P ²⁷³	Saudi	96-h LC50 19.5 mg/l	S	—	pH 6.9–7.2
	Singii	96-h LC50 11.2 mg/l	S	—	
TBP ¹⁹⁷	Zebrafish	96-h LC50 11.4 mg/l	S	25	pH 7.3–8.5 HD 100
1,2,3,5-Tetrachlorobenzene ²⁶¹	Bluegill	96-h LC50 6.4 mg/l	S	22	pH 6.7–7.8 HD 32–48
1,2,4,5-Tetrachlorobenzene ²⁶¹	Bluegill	96-h LC50 1.6 mg/l	S	22	pH 6.7–7.8 HD 32–48
1,1,1,2-Tetrachloroethane ²⁶¹	Bluegill	96-h LC50 20 mg/l	S	22	pH 6.7–7.8 HD 32–48
1,1,2,2-Tetrachloroethane ²⁶¹	Bluegill	96-h LC50 21 mg/l	S	22	pH 6.7–7.8 HD 32–48
Tetrachloroethylene ²⁶¹	Bluegill	96-h LC50 13 mg/l	S	22	pH 6.7–7.8 HD 32–48
2,3,4,6-Tetrachlorophenol ²⁶¹	Bluegill	96-h LC50 0.14 mg/l	S	22	pH 6.7–7.8 HD 32–48
2,3,5,6-Tetrachlorophenol ²⁶¹	Bluegill	96-h LC50 0.17 mg/l	S	22	pH 6.7–7.8 HD 32–48
Tetrachlorophthalide ¹⁰⁸	Carp	48-h LC50 >40 mg/l	—	—	—
	Goldfish	48-h LC50 >40 mg/l	—	—	—
	Medaka	48-h LC50 >40 mg/l	—	—	—
	Pond loach	48-h LC50 >40 mg/l	—	—	—
Thalious sulfate ²⁶¹	Bluegill	96-h LC50 120 mg/l	S	22	pH 6.7–7.8 HD 32–48
Thioacetamide ²⁶²	Fathead minnow	96-h LC50 >600 mg/l	S	22	pH 7.2–7.9 HD 40–48
Thiocarbamide ²⁶²	Fathead minnow	96-h LC50 >600 mg/l	S	22	pH 7.2–7.9 HD 40–48
Thiosemicarbazide ²⁶²	Fathead minnow	96-h LC50 20.8 mg/l	S	22	pH 7.2–7.9 HD 40–48
Thiourea ²⁶²	Fathead minnow	96-h LC50 >600 mg/l	S	22	pH 7.2–7.9 HD 40–48
Toluene ²⁶¹	Bluegill	96-h LC50 13 mg/l	S	22	pH 6.7–7.8 HD 32–48
Toluene-2,4 diisocyanate ²⁶²	Fathead minnow	96-h LC50 165 mg/l	S	22	pH 7.2–7.9 HD 40–48

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Toxaphene ²⁶	Bluegill	96-h LC50	2.6 µg/l	S	20 pH 7.4
		96-h LC50	2.4 µg/l	S	25 HD 40
		96-h LC50	4.7 µg/l	F	20 pH 7.4
		34-d LC50	0.7 µg/l	F	20 HD 272
		96-h LC50	3.4 µg/l	F	25
		7-d LC50	1.4 µg/l	F	25
	Channel catfish (<i>Ictalurus punctatus</i>)				
	Yolk-sac larvae	96-h LC50	8.0 µg/l	S	25 pH 7.4
	Swim-up larvae	96-h LC50	0.8 µg/l	S	25 HD 40
	Fingerlings	96-h LC50	2.8 µg/l	S	25
	Fingerlings	96-h LC50	5.5–7.5 µg/l	F	20–25
	Fingerlings	12-d LC50	3.7 µg/l	F	25
	Fingerlings	29-d LC50	1.9 µg/l	F	20
	Fingerlings	96-h LC50	2.7–4.7 µg/l	S	15–25 pH 6.5–8.3 HD 10–320
	Fathead minnow	96-h LC50	20 µg/l	S	20 pH 7.4
		96-h LC50	23 µg/l	S	25 HD 40
		96-h LC50	7.0 µg/l	F	20 pH 7.4
		24-d LC50	2.6 µg/l	F	20 HD 272
		96-h LC50	5.0 µg/l	F	25
		16-d LC50	1.5 µg/l	F	25
TPN (tetrachloroisophthalonitrile) ¹⁰⁰	Carp	48-h LC50	0.11 mg/l	—	—
	Goldfish	48-h LC50	0.17 mg/l	—	—
	Medaka	48-h LC50	0.09 mg/l	—	—
	Pond loach	48-h LC50	0.15 mg/l	—	—
2,4,6-Tribromophenol ²⁶⁰	Fathead minnow	48-h LC50	10 mg/l	S	22–25 pH 7.6–8.4
		96-h LC50	6.5–6.8 mg/l	F	25 HD 43–49
		192-h LC50	4.5–4.9 mg/l	F	25
Trichlorfon (formulation) ⁹⁷	Cutthroat trout	96-h LC50	3.2 mg/l	S	12 pH 7.5 HD 40
Trichlorfon (technical) ⁹⁷	Cutthroat trout	96-h LC50	4.8 mg/l	S	12 pH 6.5 HD 40
		96-h LC50	5.8 mg/l	S	7 pH 7.5
		96-h LC50	1.7 mg/l	S	12 HD 40
		96-h LC50	0.38 mg/l	S	12 pH 8.5 HD 40
		96-h LC50	0.62 mg/l	S	12 pH 7.8 HD 320
1,2,4-Trichlorobenzene ²⁶¹	Bluegill	96-h LC50	3.4 mg/l	S	22 pH 6.7–7.8 HD 32–48
1,1,1-Trichloroethane ²⁶¹	Bluegill	96-h LC50	72 mg/l	S	22 pH 6.7–7.8 HD 32–48
1,1,2-Trichloroethane ²⁶¹	Bluegill	96-h LC50	40 mg/l	S	22 pH 6.7–7.8 HD 32–48
1,1,2-Trichloroethane ²⁷¹	Guppy	24-h LC50	72 mg/l	—	— pH 8.0
		7-d LC50	70 mg/l	—	—
	Adult	24-h LC50	85 mg/l	—	—
		7-d LC50	75 mg/l	—	—

(Table 1 Continued)

Table 1—(Continued)

Pollutant	Species	Results	Exposure type	Temp. (°C)	Test conditions
Trichloroethylene ²⁸¹	Bluegill	96-h LC50 45 mg/l	S	22	pH 6.7–7.8 HD 32–48
2,4,5-Trichlorophenol ²⁸¹	Bluegill	96-h LC50 0.45 mg/l	S	22	pH 6.7–7.8 HD 32–48
2,4,6-Trichlorophenol ²⁸¹	Bluegill	96-h LC50 0.32 mg/l	S	22	pH 6.7–7.8 HD 32–48
2,4,6-Trichlorophenol ²⁸⁰	Fathead minnow	48-h LC50 7.7 mg/l 96-h LC50 8.6–9.7 mg/l 192-h LC50 5.8–6.4 mg/l	S F F	22–25 25 25	HD 43–49
Trifluralin ¹⁰⁸	Carp	48-h LC50 1.0 mg/l	—	—	—
	Goldfish	48-h LC50 0.85 mg/l	—	—	—
	Medaka	48-h LC50 0.43 mg/l	—	—	—
	Pond loach	48-h LC50 0.35 mg/l	—	—	—
2,4,6-Trinitrophenol ²⁸¹	Bluegill	96-h LC50 170 mg/l	S	22	pH 6.7–7.8 HD 32–48
Zinc ¹⁸³	Rainbow trout Juvenile	12-d LC50 0.41 mg/l	F	8	HD 20–52
Zinc ¹⁸⁰	<i>Puntius sophore</i>	96-h LC50 31.2 mg/l	S	21	pH 7.4
	<i>Rasbora daniconius neilgeriensis</i>	96-h LC50 35.8 mg/l	S	21	HD 260
	Saudi	96-h LC50 29.9 mg/l	S	21	
	Guppy	96-h LC50 55 mg/l	S	21	
Zinc sulfate ²²²	Guppy	96-h LC50 1.7 mg/l 96-h LC50 1.5 mg/l	S F	25 25	pH 7.2 HD 30
	Adult	96-h LC50 5.1–6.4 mg/l	S	25	
Zineb ¹⁰⁸	Carp	48-h LC50 >40 mg/l	—	—	—
	Goldfish	48-h LC50 >40 mg/l	—	—	—
	Medaka	48-h LC50 >40 mg/l	—	—	—
	Pond loach	48-h LC50 >40 mg/l	—	—	—

* S = static bioassay

b HD = hardness, mg/l

c MATC = maximum acceptable toxicant concentration

d F = flow-through bioassay

e R = renewal bioassay

f BFC = bioconcentration factor

g IP = intraperitoneal injection

Sailatha *et al.*¹⁰¹ The 48-hour LC50 values for technical grade and commercial grade malathion was 5.6 and 0.38 mg/l, respectively. Gopal *et al.*¹⁰² tested the acute toxicity of endosulfan to an insect nymph, a tadpole, and a catfish (*Clarias batrachus*). The tadpole (*Rana tigrina*) was the most sensitive species and the insect (*Enallagma* sp.) was the most resistant. The 96-hour LC50 value of endosulfan for the catfish was 14 µg/l. The acute toxicity of endosulfan, dichlorvos (DDVP), and carbofuran to *Mystus vittatus* and *Ophiocephalus punctatus* was determined by Verma *et al.*¹⁰³ To both species of fish, endosulfan was the most toxic and DDVP was the least toxic. *Mystus vittatus* was the more sensitive species to endosulfan and DDVP. The 96-hour LC50 values for *Mystus vittatus* ranged from 0.79 to 2 040 µg/l for endosulfan and DDVP, respectively. Devi *et al.*¹⁰⁴ reported the acute toxicity and bioaccumulation of technical grade, formulation, and two

isomers of endosulfan to *Channa punctata*. The 96-hour LC50 values ranged from 0.16 µg/l for isomer A to 16 µg/l for the 4% dust formulation. Endosulfan and both isomers were found in the liver, kidney, gills, and brain of fish exposed to 6.5 µg/l of technical grade endosulfan. Endosulfan lactone and endosulfan alcohol were also found in these tissues. Endosulfan hydroxyether was found in kidney, gills, and liver while endosulfan ether was found in the liver and kidney. Nagarathnamma and Ramamurthi¹⁰⁵ reported the acute toxicity of methyl parathion to four invertebrate species and the carp. The 48-hour LC50 values ranged from 1 to 40 mg/l with the carp having an intermediate value of 12 mg/l. The acute toxicity of four carbamate pesticides to three species of fish was determined by Hejduk and Svobodová.¹⁰⁶ Rainbow trout was the most sensitive species to mancozeb, dioxacarb, and benomyl, and the guppy was the most sensitive to carbofuran.

The carp was the most resistant species to three of the pesticides. Scirocchi and D'Erme¹⁰⁷ tested the acute toxicity of seven insecticides to six species of fish. At concentrations of insecticides that caused 100% mortality of mosquito larvae, only Abate had no effect on all six fish species. Hashimoto and Nishiuchi¹⁰⁸ established methods to evaluate the acute toxicity of pesticides to aquatic life and used these methods to assess the acute toxicity of 42 registered pesticides. Toxicity tests with 32 pesticides were conducted to determine their effects on fish, aquatic invertebrates, and a tadpole. Tests were also conducted to investigate the effects of temperature, formulation, and method of application.

Bansal *et al.*¹⁰⁹ conducted 30-day tests with chlordane, carbaryl, and methyl demeton using larvae of four species of carp to develop information for predicting long-term effects from acute toxicity tests. Standing crop (total weight of live fish divided by the original number of exposed larvae) was used as the measure of effect for the 30-day exposure. The authors recommended that the ratio between standing crop values and 96-hour LC50 values be used by multiplying this ratio by the 96-hour LC50 value of another species to estimate long-term effects for that species. Acute tests and life cycle chronic toxicity studies of kepone and mirex using the fathead minnow were conducted by Buckler *et al.*¹¹⁰ The MATC for kepone was between 1.2 and 3.1 $\mu\text{g/l}$ and was based on survival and growth. Mirex at concentrations in excess of water solubility (50 to 60 $\mu\text{g/l}$) was not lethal to fathead minnows in 60-day exposures. In the life cycle chronic exposure to mirex, there were no significant adverse effects in the high concentration of 34 $\mu\text{g/l}$. The BCF for kepone was 1 100 to 2 200 and for mirex was 12 000 to 28 000. The organization and adaptation of laboratory aquatic ecosystems to resource availability, exploitation, and dieldrin were examined by Woltering.¹¹¹ In general, dieldrin reduced the growth and reproduction of guppies. With 0% exploitation, the ecosystem responded immediately with apparent dieldrin-induced mortalities of mature fish, however, the population recovered its lost biomass. There was no apparent initial response at 40% exploitation, but after 15 months the guppy population became extinct.

Tsuge *et al.*¹¹² exposed the guppy, red snail, mosquito larva, and *Daphnia pulex* to 1 $\mu\text{g/l}$ of each of five [¹⁴C]-labelled pesticides. Uptake of DDT was most rapid with mosquito larvae. Aldrin was rapidly transformed to dieldrin by the guppy. Prothiophos was rapidly degraded by the guppy. The uptake of isothioate, carbaryl, and XMC was low, and these pesticides were metabolized rapidly by all species. Ratnakar *et al.*¹¹³ developed and used a colorimetric method to measure residues of BHC in fish tissue. The livers of 10 fish exposed to 100 mg/l contained residues of 146 mg of BHC. The ability of sediments to degrade kepone or mirex and the accumulation, metabolism, and elimination of kepone or mirex by the bluegill were studied by Skaar *et al.*¹¹⁴ No direct evidence of degradation of either compound was found after 56 days of incubation in sediment under aerobic or anaerobic conditions. After 28 days of exposure of bluegills to [¹⁴C]-labelled kepone or mirex in water and natural food, the fish accumulated kepone and mirex equal to 10 606 and 12 274 times the respective water concentrations. Neither compound was metabolized by bluegill. The accumulation, retention, and elimination of mirex by brook trout fed a diet of 0.7 μg mirex/g body weight were examined by Skea *et al.*¹¹⁵ The rate of accumulation was

rapid and never reached a plateau. At the end of the feeding period, the average concentration of mirex was 6.3 $\mu\text{g/g}$ and this concentration declined to 2.1 $\mu\text{g/g}$ at the end of the 385-day period of mirex-free food. However, during the elimination phase, the fish increased in size from an average of 175 g to 571 g. Thus, the decline in concentration of mirex was attributed to growth dilution. The uptake, metabolism, and elimination of diazinon by rainbow trout, carp, loach (*Misgurnus anguillicaudatus*), and shrimp were measured by Seguchi and Asaka.¹¹⁶ Equilibrium concentrations were reached in 3 days in fish exposed to 18 $\mu\text{g/l}$ of diazinon for 14 days. The metabolite M-6 was found in all three species, but M-2 was found only in rainbow trout and carp. When the fish were returned to clean water, diazinon and its metabolites were eliminated rapidly. A linear relationship was found for the BCF and the fat content of the fish. Gray and Knowles¹¹⁷ studied the metabolism and elimination of propamocarb-¹⁴C injected into bluegill and channel catfish. Elimination of injected radiocarbon was rapid for both species. With channel catfish, most of the radio carbon in the water partitioned into methylene chloride, whereas, with the bluegill most of the radiocarbon was water-soluble. Glickman *et al.*¹¹⁸ investigated the distribution and metabolism of cis- and trans-permethrin isomers in rainbow trout to evaluate the differential toxicity of permethrin to fish and mammals. The 24-hour LC50 values for permethrin, cis-permethrin, and trans-permethrin were 18, 25, and 14 $\mu\text{g/l}$, respectively. Both [¹⁴C]-permethrin isomers were readily taken up and eliminated by trout. High concentrations of a polar metabolite were found in the bile within 4 hours of exposure. The relative absence of permethrin hydrolysis products in the bile and the small amount of radioactivity excreted in urine indicated that the ability of trout to hydrolyze permethrin, *in vivo*, was small. In a companion study, Glickman and Lech¹¹⁹ compared the rate of hydrolysis of cis- and trans-permethrin by rainbow trout and mouse tissue *in vitro*. Mouse liver, kidney, and plasma incubated at 37°C hydrolyzed trans[¹⁴C]-permethrin approximately 168, 38, and 59 times faster, respectively, than these tissues in fish incubated at 12°C. The results suggested that rainbow trout tissues have a lower capacity to hydrolyze permethrin, which may explain the relative absence of permethrin hydrolytic metabolites in trout exposed to either cis- or trans-permethrin.

The *in vitro* metabolism of carbofuran by liver microsomes of the padfield fish, sepat siam (*Trichogaster pectoralis*) was analyzed by Gill.¹²⁰ The pesticide was metabolized by the microsomal-NADPH system to give two major metabolic products, N-hydroxymethyl carbofuran and 3-hydroxy-carbofuran, and two minor products, 3-keto-carbofuran and 3-keto-carbofuran phenol. The results showed that oxidative hydroxylation was the major route of metabolism. Sudershan and Khan¹²¹ exposed bluegills for 48 hours in a static system to 5 $\mu\text{g/l}$ each of cis[¹⁴C]chlordane and cis[¹⁴C]photochlordane to investigate the metabolic fate of cis[¹⁴C]photochlordane. Absorption by the fish was rapid as more than 95% of the radioactivity was lost from the experimental jars. Elimination of chlordane was linear with about 20% being eliminated during the 6-week post-exposure period. Elimination of photochlordane was biphasic, being rapid in the early phase. Less than 7% of the retained radioactivity in the chlordane-treated fish was in the form of two conjugates which on hydrolysis gave at least eight hydroxylated products. For photochlordane,

16% of the retained radioactivity was in the form of nine apolar and five polar metabolites. In a similar study,¹²² bluegill exposed to 50 µg/l of [¹⁴C]dieldrin absorbed 73% of the radioactivity in 48 hours. After transfer to clean water, only 16% of the absorbed radioactivity was eliminated in 23 days. Of the absorbed radioactivity recovered from the fish, nine radioactive spots were isolated, which included unchanged dieldrin (74%) and the two major metabolites pentachloroketone (8.2%) and aldrin-trans-diol (8%). The same authors¹²³ evaluated the uptake, metabolism, and elimination of [¹⁴C]endrin by bluegills. Bluegills absorbed 85% of 1 µg/l of endrin from water within 48 hours. The absorbed radiocarbon was eliminated linearly and had a half-life of about 4 weeks. Analysis of this eliminated radioactivity gave only conjugated metabolites. Seven radioactive metabolites were found but only 12-anti-hydroxyendrin and 12-syn-hydroxyendrin were tentatively identified. These two metabolites were also present as conjugates in the organs of the fish. Seventy-three percent of the radioactivity recovered from fish extracts was in the form of unchanged endrin.

Rao *et al.*¹²⁴ conducted tests with the fish *Macroglythys aculeatum* to study the toxicity and metabolism of endosulfan. The 96-hour LC50 value was 3.5 µg/l. Endosulfan was metabolized to endosulfan sulfate in the brain, gill, gut, liver, and kidney of fish that survived 96-hour exposure to 0.5 µg/l. At both lethal and sublethal concentrations of endosulfan, O₂ consumption and total nitrogen excretion of the fish was decreased as compared to control fish. Denison *et al.*¹²⁵ studied the vitellogenin-like protein (VLP) and binding of DDT in the serum of insecticide-resistant mosquitofish. Serum from insecticide-resistant fish contained VLP year round. *In vitro* incubation of [p,p'-³H]DDT with reproductive or non-reproductive resistant and susceptible fish serum, lead to its association with three proteins (VLP, a low molecular weight lipoprotein, and albumin). The authors suggested that estrogenic and/or proestrogenic xenobiotics from the environment of the fish induced the formation of VLP which can bind and transport insecticides during the non-productive season. Leatherland and Sonstegard¹²⁶ examined the effects of dietary mirex and/or PCBs on Ca²⁺ and Mg²⁺ homeostasis in rainbow trout and coho salmon (*Oncorhynchus kisutch*) and measured serum Ca²⁺ and Mg²⁺ in feral coho salmon from Lakes Ontario, Erie, and Michigan. Dietary mirex (50 mg/kg body weight) or PCBs (500 mg/kg body weight) significantly lowered serum Ca²⁺ in coho salmon. Lower dietary levels and a mixture diet of mirex and PCBs were without effect. There were no significant changes in serum Mg²⁺ concentrations. None of the contaminated diets had a significant effect on Ca²⁺ or Mg²⁺ levels in serum, muscle, or skeleton of rainbow trout. The hypocalcaemia of feral coho salmon from Lake Erie could not be related to organochlorine content of the fish but may have been a factor in the poor growth of coho salmon in Lake Erie as compared with those in Lakes Ontario and Michigan. Bano *et al.*¹²⁷ fed catfish (*Clarias batrachus*) 1, 2, 5, 10, and 25 mg DDT/g body weight and sampled the fish at 12-, 48-, and 120-hour intervals for measurements of muscle constituents. All fish fed 25 mg/g died within 24 hours but there was no mortality in lower concentrations. At the high sublethal dose of 10 mg/g, fish showed a significant decrease in crude protein, pure protein, albumin, total water soluble nitrogen, and water soluble nitrogen.

The acute effects of methyl parathion on carbohydrate metabolism of the Indian catfish (*Heteropneustes fossilis*) was measured by Srivastava and Singh.¹²⁸ Fish were exposed to 5.6 mg/l (80% of 96-hour LC50) for 3, 6, 12, 48, and 96 hours. Muscle glycogen levels were significantly less at all exposure times and liver glycogen was less at 6 hours but greater at 12 hours. Blood glucose levels were significantly higher at 3 and 6 hours; blood pyruvate levels were higher at 3, 12, and 96 hours; and blood lactate levels were significantly greater at 48 and 96 hours. In another body,¹²⁹ aldrin was more toxic than parathion to the Indian catfish. Fish that were exposed to a sublethal concentration of each compound and fish that survived the 96-hour LC50 concentration of each pesticide had reduced ³²P uptake and a significantly decreased level of total gonadotrophin in the pituitary gland and blood serum. Measurements of muscle and liver glycogen, and blood glucose, pyruvate, and lactate from this catfish were made at 3-, 6-, 12-, 48, and 96-hour exposures with mixtures of aldrin and formothion.¹³⁰ All parameters were significantly different than control values at various exposure times. Muscle glycogen was decreased at 6 and 48 hours, and hepatic glycogen was decreased at 3-, 6-, 12-, and 48-hour exposures. Blood glucose levels were decreased at 3, 12, 48, and 96 hours; blood pyruvate was decreased at 3 hours; and blood lactate levels were increased at 3, 6, 12, and 96 hours. Singh and Singh¹³¹ exposed the Indian catfish, in a similar study, to two sublethal concentrations of malathion and endrin and measured testicular ³²P uptake, gonadotrophin level, lipid and cholesterol content of testis, liver, and blood serum. In fish exposed for 4 weeks to both concentrations of each pesticide, there was a significant decrease in testicular ³²P uptake and a significant increase in level of total lipid of liver and cholesterol content of testis, liver, and blood serum.

The effects of sumithion on O₂ consumption, succinate dehydrogenase and acetylcholinesterase (AChE) of brain, gill, liver, and muscle of *Etroplus maculatus* were measured by Vijayalakshmi.¹³² For all four tissues, there was a significant decrease in O₂ consumption, succinate dehydrogenase activity, and AChE activity. Sahib *et al.*¹³³ exposed the mozambique mouthbrooder (*Tilapia mozsambica*) to 2 mg/l of malathion for 48 hours and measured body weight, body water content, O₂ consumption of the fish, and the level of sodium (Na), potassium (K), and calcium (Ca) in muscle, gill, and liver. There was no effect on body weight or body water content. Oxygen consumption was significantly greater after 12- and 24-hour exposures to malathion but was not different than that of control fish at 36 and 48 hours. Sodium and K concentrations in muscle, gill, and liver of exposed fish was significantly less than that of control fish. Calcium concentration in only the gill of exposed fish was significantly less than that of control fish. In a similar study,¹³⁴ ammonia and glutamate dehydrogenase showed a general decrease with time of exposure of this species to malathion. Free amino acid of the muscle, gill, and liver of fish exposed to malathion for 48 hours was significantly greater than control values. Dalela *et al.*¹³⁵ evaluated the effects of lethal and sublethal concentrations of chlordane, Metasystox, and carbaryl on O₂ consumption of *Saccobranchius fossilis*. All-or-none responses were observed at LC50 concentrations of each pesticide. At lower concentrations, O₂ consumption was characterized by three distinct phases, (a) phase of sensitization, (b) responsive phase, and

(c) the phase of normalization. Rath and Misra¹³⁶ measured the effects of exposure to 0.5 mg/l of DDVP on the O₂ consumption of gill, brain, and muscle tissue of *Tilapia mossambica* of different ages. Oxygen consumption of all tissues of all age groups inhibited when exposed to DDVP for 1, 2, 3, and 4 weeks. The gill had a greater inhibition than muscle or brain tissue. When fish were returned to clean water the recovery rate of O₂ consumption of muscle was greater than that of brain and gill tissue and was inversely related to age. Tests on the effects of DDVP on brain and liver AChE activity and pigment dispersion of the mozambique mouthbrooder,^{137,138} indicated that there was a significant negative correlation between body weight and AChE activity of fish exposed to the insecticide. The percent of enzyme inhibition had a positive correlation with DDVP concentration and time of exposure. The AChE activity of small fish was more sensitive to DDVP than that of larger fish. A concentration of 1 mg/l of DDVP caused dispersed chromatophors. After transfer to clean water the exposed fish gradually regained their original color showing chromatic aggregation within 96 hours. Verma *et al.*¹³⁹ investigated the effects of 30-day exposure to thiotox, DDVP, and carbofuran and combinations of these pesticides on serum transaminases in the African catfish (*Mystus vittatus*). Alterations were greater in serum glutamic oxaloacetic transaminase than in serum glutamic pyruvic transaminase. Significant stimulation was found except at the lowest concentrations. The hematological alterations of *Mystus vittatus* following exposure to thiotox, DDVP, and carbofuran pesticides and their combinations at one-fifth, one-tenth, and one-fifteenth of the 96-hour LC50 values were observed by Dalela *et al.*¹⁴⁰ Concentrations of glucose, lactate, total P, non-protein nitrogen, Na, K, magnesium (Mg), Ca, iron (Fe), and Cl⁻ concentrations increased with pesticide concentration both singly and in combination, whereas protein and cholesterol concentrations decreased. Alterations of protein, K, Mg, and Ca were insignificant while all other components were significantly altered.

Verma *et al.*¹⁴¹ investigated the role of ascorbic acid on some hematological parameters of *Saccobranchius fossilis* exposed to thiotox (35% EC) and malathion (50% EC). Fish fed a diet with and without added ascorbic acid were exposed for 15 and 30 days to 2 µg/l of thiotox and 5.2 mg/l of malathion. Exposure to both pesticides for 30 days induced significant alterations in the blood parameters of the fish not fed added ascorbic acid while the fish fed added ascorbic acid did not have any significant alterations of blood parameters. Shaffi¹⁴² measured the effects of 4-hour exposures to thiodon on kidney, brain, liver, and muscle non-specific phosphomonoesterases from nine species of fish. Exposure to sublethal concentrations (5 and 10 mg/l) and lethal concentrations (15 and 20 mg/l) caused an increase and decrease, respectively, of alkaline phosphatase in all four tissues. The reverse was found for acid phosphatase. The ultrastructural morphology of intestinal cells of goldfish (*Carassius auratus*) exposed to toxaphene was examined by Caceci.¹⁴³ The intestinal cells of fish exposed to toxaphene showed a marked development of very osmiophilic whorled structures that were derived from mitochondria and other membranous intracellular components. The hypothesis that they may constitute a temporary emergency storage form of intracellular lipids was presented. Sastry and Sharma¹⁴⁴ exposed *Ophiocephalus punctatus* to 0.2 and 0.4 mg/l of dia-

zinon for 30 days and studied the histopathological changes in the liver, intestine, kidney, and gills and in 16 hematological parameters. Liver damage of fish exposed for 30 days was characterized by fibrosis, small degenerated nuclei, and invasion of a number of phagocytes into the necrosed tissue. A few intestinal villi were necrosed and nuclei were reduced in volume. Shrinkage of the glomerular network and necrosis of proximal tubules were the most conspicuous changes in the kidney. There was no significant effect on the hematological parameters of fish exposed to 0.2 mg/l for 15 or 30 days. For fish exposed to 0.4 mg/l, only glucose at 15 days and hemoglobin at 30 days were significantly different than that of control fish.

The effects of atrazine, lindane, and ethyl parathion on the olfactory bulb response of rainbow trout were measured by Borie *et al.*¹⁴⁵ Nasal infusions of 1 000, 10, 0.1, and 0.01 µg/l of each pesticide produced characteristic amplitude and frequency changes. Kling¹⁴⁶ investigated the effects of Lebaycid (fenthion) on the ovaries and reproductive activity of *Tilapia leucosticta*. Periodic exposure to sublethal concentrations of 4 and 7 mg/l caused total atresia of the ovaries in 90% of the individuals. The gonads were filled with atretic follicles, and mature eggs were not found. Females exposed to Labaycid were unable to spawn for at least 9 weeks after exposure, whereas the spawning period of control fish was 5 weeks.

Herbicides. Geen *et al.*¹⁴⁷ examined the fate and toxicity of acephate (Orthene) added to Hidden Creek, a tributary of the Fraser River in British Columbia. An acephate concentration of 1 100 to 1 200 µg/l for 5 hours caused no fish (yearling rainbow trout) or insect mortality but was rapidly taken up in the animals and the sediments. Both acephate and its conversion product, methamidophos, declined quickly in tissue and water after exposure was stopped indicating the limited persistence of the chemicals in this stream. Aaronson *et al.*¹⁴⁸ investigated a fish kill in a pond in which the farmland surrounding the pond had received recent application of Counter (an organophosphate), Sutan (a thiocarbamate), and Bladex (a triazine). None of these pesticides were identified in a sample of water from the pond, however, the herbicide atrazine was positively identified and confirmed. The origin of atrazine was unknown.

Jirásek *et al.*¹⁴⁹ determined the acute toxicity of three herbicides to carp, crucian carp (*Carassius carassius*), guppy, *Tinca tinca*, and *Leucaspis delinatus*. Both Lasso and Bidisin were toxic to fish at approximately 0.01 ml/l, except for the crucian carp. TCA was not found to be toxic to any of these species. The toxicity of Lasso and Bidisin, to four species of fish was examined by Kepr and Adámek.¹⁵⁰ Both compounds were highly toxic and the clinical and pathological pattern of intoxication were described. The acute toxicity of 2,4-D to three species of cyprinidae was determined by Vardia and Durve.¹⁵¹ The 96-hour LC50 values of 2,4-D to *Labeo boga*, *Rasbora neilgherriensis*, and the carp were 3.8, 5.6, and 20 mg/l, respectively. The effect of temperature on the acute toxicity of 2,4-D to carp was reported by Vardia and Durve.¹⁵² The 96-hour LC50 values for 2,4-D at 39°, 33°, 29°, 25°, 20°, and 17°C were 5.1, 15, 20, 24, 31, and 35 mg/l, respectively. Rand and Barthalmus¹⁵³ used an unsignalled continuous avoidance technique to evaluate the subacute effects of 15.4 mg/l of 2,4-D on the conditioned avoidance learning in goldfish. Goldfish exposed for 24 hours on the ninth day of conditioning

had no significant differences in response against controls, while fish exposed on the first day of conditioning exhibited differences in the magnitude of avoidance responding. Significant differences in pattern, rate of acquisition, and maintenance of the avoidance baseline were seen in fish exposed for 2 weeks. Finlayson¹⁵⁴ conducted flow-through acute toxicity tests of two herbicides to golden shiners (*Notemigonus crysoleucas*). Komeen (copper-ethylenediamine complex) was more toxic in soft water than in hard water. Hydrothol-191 (mono-N,N-dimethylakylamine salt of Endothal) was more toxic in hard water. The acute effects of the herbicide, Machete, on various life stages of the carp were examined by Cha and Kim.¹⁵⁵ The 24- and 48-hour LC50 values to the prelarvae were 0.86 and 0.43 mg/l, respectively. For juveniles, the 24-, 48-, 96-hour, and incipient LC50 values were 0.625, 0.365, 0.180, and 0.135 mg/l, respectively. Embryos were the most resistant life stage.

The effect of the herbicide, dinoseb acetate, on mortality and on muscle proteins of the guppy was reported by Paulov.¹⁵⁶ All fish died at 0.8 mg/l. Polyacrylamide gel electrophoresis revealed a pronounced qualitative change in muscle proteograms. Gunkel and Streitz¹⁵⁷ conducted studies to examine the mechanisms of bioaccumulation of atrazine in a mollusk (*Ancylus fluviatilis*) and whitefish (*Coregonus fera*). Measurements were made with non-labelled and [¹⁴C]ring-labelled atrazine as well as tritiated water. The pattern of atrazine accumulation by the mollusk was similar to that of the fish. The maximum accumulation in the whitefish occurred in about 1 day in a 24-day exposure to 250 µg/l of atrazine. The kinetics of accumulation indicated that atrazine was accumulated via the gills and that other organs were contaminated via the blood. The BCF for various organs and tissues ranged from 1.7 for blood to 48.5 for gall bladder while the BCF for whole fish was 2.8. Kubota *et al.*¹⁵⁸ exposed carp to 1.5 mg/l of benthocarb for 21 days and reported the ultrastructure of inclusion-like bodies in the hepatocytes. The inclusion-like bodies consisted of rough endoplasmic reticulum that surrounded fat droplets. The authors suggested that fat droplets may contain benthocarb and that the inclusion-like bodies may be a form of intracellular defense.

Piscicides and anesthetics. The ecological impact of lampricide treatment on sea lamprey (*Petromyzon marinus*) ammocoetes and transformed individuals in streams of the upper Great Lakes was described by Torblaa and Westman.¹⁵⁹ Sea lamprey ammocoetes were found in fewer locations after treatment than before treatment began. After treatment, the abundance of ammocoetes and transformed individuals declined in most watersheds. Increased growth after treatment was related to reduced density. The sex ratio of larval and metamorphosing populations were highly variable during initial lampricide treatment. The shift to femaleness in larval populations was found to cause a similar shift in adult sea lamprey populations in the upper Great Lakes. The effects of weirs and chemical treatment to control the sea lamprey on fish populations of the upper Great Lakes was discussed by Dahl and McDonald.¹⁶⁰ Fish kills of native species have been common but the information on migratory species showed no large-scale detrimental effects. The only reported damage to the resident stream species was the near loss of the stonecat (*Noturus flavus*) from tributaries to the southwest corner of Lake Superior because of chemical treatment. The authors concluded that as long as

existing control measures continue migratory and resident fish populations will be affected. The development of relative selective larvicides to control sea lamprey in the Great Lakes was discussed by Howell *et al.*¹⁶¹ Fifteen halogenated mononitrophenol and 56 substituted nitrosalicylanilid compounds have been found to be selectively toxic to larval lamprey. The major toxicant used in sea lamprey control was found to be 3-trifluoromethyl-4-nitrophenol (TFM) while 5,2'-dichloro-4'-nitrosalicylanilide (Bayer 73) was used as an additive to increase the toxicity of TFM. The effects of water quality, pharmacology, and selective toxicity of the two compounds were discussed.

Marking and Bills¹⁶² determined the acute toxicity of four registered or candidate fish toxicants to bighead carp (*Aristichthys nobilis*), silver carp (*Hypophthalmichthys molitrix*), grass carp (*Ctenopharyngodon idella*), and carp (Table 1). Salicylanilide I was less toxic than antimycin but more toxic than noxfish. Toxicity of GD-174 was little affected by water temperature and water hardness but was more toxic at higher pH. Schultz and Harman¹⁶³ measured the effects of 13 fishery chemicals on the *in vitro* activity of glucose-6-phosphate dehydrogenase (G6PDH). Potassium permanganate was the most inhibitory chemical. Three therapeutic chemicals (Acriflavine, Hyamine 1622, and Hyamine 3500) were about 5 times as inhibitory as the piscicides, anesthetics, and formalin. The authors concluded that the *in vitro* inhibition of G6PDH could not be used to predict toxicity of the fishery chemicals.

Ferreira *et al.*¹⁶⁴ studied the effects of anesthesia with benzocaine hydrochloride (BH) and neutralized benzocaine hydrochloride (NBH) on hematology in the carp. Significant correlations were found between BH concentrations and blood PO₂, Hb, ATP, plasma Na, K, Cl, Osm, and induction times. Some significant interrelationships between NBH and several whole blood parameters were recorded. In general, effects of BH and NBH on the blood agreed with the results obtained from MS222 and neutralized MS222 in other studies. Rainbow trout were exposed by Hlavcek and Bulkeley¹⁶⁵ to malachite green to determine if this therapeutic agent caused chronic leucopaenia. Fish exposed to 1.35, 13.5, or 21 mg/l for 25 to 30 minutes had major changes in blood counts during the first 24 hours. Recovery was essentially completed in 1 to 4 days post-exposure. Thrombocytosis developed during the first 24 hours in fish exposed to 42 or 72 mg/l for 5 minutes. Lymphopaenia and neutrophilia also developed but abated 4 days post-treatment. The authors concluded that because leucocyte changes in the exposed fish also occurred in controls, the leucocyte changes were the result of a non-specific stress syndrome.

Inorganics. Fish from 98 monitoring stations in the U. S. were collected during 1976 to 1977 and analyzed for Cd, lead (Pb), mercury (Hg), arsenic (As), and selenium (Se).¹⁶⁶ Ranges and geometric mean values in mg/kg wet weight were: Cd, 0.01 to 1.04, 0.07; Pb, 0.10 to 4.92, 0.32; Hg, 0.01 to 0.84, 0.11; As, 0.05 to 2.92, 0.27; and Se, 0.05 to 2.87, 0.56. Significant temporal decreases in whole-body concentrations were found for Cd and Hg. There was an increase in As and no change in Pb or Se. Marks *et al.*¹⁶⁷ studied nine heavy metals in five species of fish (*Mugil cephalus*, *Aldrichetta fosteri*, *Amniataba caudavittatus*, *Acanthopagrus butcheri*, and *Nematalosa vlaminhi*) in a river in Australia. Iron, zinc (Zn), copper (Cu), and manganese (Mn) decreased or remained

constant with increasing body weight especially for fish weighing more than 50 g. For longer fish metal concentrations were thought to approximate levels found in fish in unpolluted streams. Metal concentrations were higher in fish ingesting bottom sediments than those with other diets. Cadmium, Cu, and Hg concentrations in the livers of northern pike (*Esox lucius*) and white sucker (*Catostomus commersoni*) from five Canadian lakes were measured by McFarlane and Franzin.¹⁶⁸ Metal concentrations in pike increased with age of the fish and were not consistent with the degree of contamination in the lakes. Calcium content of the lakes influenced liver metal concentrations. Paasivirta *et al.*¹⁶⁹ sampled pike in May 1980 from three areas of Lake Paijanne, Finland, and analyzed the fish for total Hg, methyl mercury, hexachlorobenzene, DDE, PCB, and chlorophenolic residues and compared the results to earlier studies. The levels of DDE ranged from a mean of 3.7 to 16 ng/g for three areas showing an increasing trend when the influence of fat was eliminated. Hexachlorobenzene was found for the first time and the mean concentrations ranged from 0.4 to 1.0 ng/g. There was a significant decrease in Hg from all three areas from 1970 to 1980. Pentachlorophenol occurred at 10 to 70 ng/g (fresh weight) at all areas.

The effects of pollution from a U/Cu mine on the diet of the purple striped gudgeon (*Mogurnda mogurnda*) were investigated by Jeffree and Williams.¹⁷⁰ Acid discharges of Cu, Zn, Mn, and sulfur (S) decreased the abundance of aquatic macrofaunal species. However, conclusive predictions in dietary changes based on the field work in this study were difficult because of the complexity factors and the lack of experimental observations to support the phenomena observed in the field. The distribution of several heavy metals and radionuclides in the water, sediments, and lake trout (*Salvelinus namaycush*) near an operating Hg mine and abandoned U mine were determined by Moore and Sutherland.¹⁷¹ Concentrations of Hg, Pb, Mg, and nickel (Ni) decreased with distance from the mines and sediment bonding did occur. Low Hg concentrations in the lake trout (0.03 mg/kg) relative to Hg water concentrations were thought to result from the low uptake rate and the ability of the fish to seek uncontaminated water. The effects of Cu, Zn, and Cd from acid mine wastes were studied in trout residing in the Sacramento River Basin by Wilson *et al.*¹⁷² No correlation was found between metal concentrations in the flesh and the water, however, liver metal concentrations increased with increasing water metal concentrations and fish age. Phillips and Gregory¹⁷³ studied the accumulation of As, Cu, Hg, Pb, Se, and Zn by northern pike reared in decant water from a surface coal mine in Montana. The Hg uptake rate was only slightly higher than the pike in natural Montana waters and there was no accumulation of the other metals from the coal mining effluent. Heavy metal concentrations in three fish, fantail darter (*Etheostoma flabellare*), blacknose dace (*Rhinichthys atratulus*), and bluehead chub (*Nocomis biguttatus*), found in Virginia streams polluted from automobile emissions were studied by VanHassel *et al.*¹⁷⁴ The concentrations of the four metals analyzed (Ni, Cd, Pb, and Zn) in the fish corresponded to traffic density along the stream with the exception of Cd in the blacknose dace and Ni in the bluehead chub and fantail darter.

Mercury concentrations in the muscle and organs of several species of fish caught in the Regnitz River (Germany) were analyzed by Pleyer.¹⁷⁵ Species of fish in similar habitats

showed considerable differences in Hg concentrations while fish from different habitats showed similar Hg content. The contrasting results were explained by the affinity of species to a given equivalent Hg content of the water. Akielaszek and Haines¹⁷⁶ studied Hg in muscle tissue of brook trout and lake trout from three wilderness lakes in order to test the hypothesis that fish inhabiting oligotrophic lakes have elevated Hg levels. The authors found this to be true and suggested that Hg accumulation in oligotrophic lakes was facilitated because there are not enough dissolved organic compounds to complex the metal resulting in greater availability of the metal to fish. The accumulation of Hg in brook sticklebacks and pearl dace in streams contaminated by dredge spoils were studied by Munro and Gummer.¹⁷⁷ Mercury concentrations in the sediments ranged up to 38 mg/kg and fish concentrations were below 0.15 mg/kg. Hildebrand *et al.*¹⁷⁸ studied Hg accumulation in axial muscles of rock bass (*Ambloplites rupestris*) and the hogsucker (*Hypentelium nigricans*) from the North Fork of the Holston River in Virginia and Tennessee. Approximately 96% of the variance in total Hg concentration in the rock bass and 80.5% of the variance in total Hg concentration in hogsuckers were directly related to Hg levels in the water.

The effects of urban runoff on Coyote Creek in California were studied by Pitt and Bozeman.¹⁷⁹ Mosquitofish were analyzed for Pb and Zn in urbanized and non-urbanized areas of the stream and taxonomic diversity was also studied. The effects of Cu from copper sulfate herbicides in ponds were studied by Bohl *et al.*¹⁸⁰ High levels of Cu were found in the sediments but low levels were found in the fish.

The chemistry, toxicity, and pathology of Al was studied in relation to stream pollution from an anodizing plant in Scotland.¹⁸¹ The acute toxicity of aluminum (Al) to fish was found to depend on pH of the river water. At neutral pH, no acute toxicity occurred at 200 mg/l but 40 and 100% mortality occurred at the same concentration at pH 8 and 9, respectively. It was suggested that the maximum concentration of Al in the river be limited to 0.5 mg/l. Baker¹⁸² studied Al toxicity to rainbow trout and white sucker as related to acid precipitation and Adirondack surface water quality. In synthetic acidified Al solutions, the toxicity of inorganic Al varied at different pH levels and with different life history stages. Complexation of Al with citrate and natural organic chelates in surface waters seemed to eliminate Al toxicity to fry. General trends in water quality in relation to relative sensitivities of life stages were discussed.

Davies and Woodling¹⁸³ compared the results of laboratory bioassays and *in situ* stream bioassays using rainbow trout, brown trout, and brook trout to determine their usefulness in predicting Cu, Cd, and Zn toxicity in polluted streams. The results showed close agreement and the metal primarily responsible for toxicity could often be identified. Knittel¹⁸⁴ studied the effects of Cd, Zn, and Cu on the susceptibility of rainbow trout to redmouth infection (*Yersinia ruckeri*). A 96-hour exposure to Zn at 98 µg/l did not decrease tolerance to infection but exposure to a near lethal level of Cd at 0.9 µg/l increased susceptibility to infection. Copper concentrations of 2.0 µg/l also increased susceptibility. Bellavere and Gorbis¹⁸⁵ conducted 96-hour static bioassays with chromium (Cr), Cd, and Cu on three species of aquatic organisms including the zebrafish (*Brachydanio rerio*). Of the three metals tested Cu was the most toxic. The 96-hour LC50 values for Cr, Cd, and

Cu were 58.5 mg/l, 4.35 mg/l, and 0.21 mg/l, respectively. The effects of acclimation to Cu on Cu and Zn toxicity in rainbow trout were determined by Dixon and Sprague.¹⁸⁶ Three-week exposures to 0.29, 0.40, and 0.59 of the incipient lethal level (300 µg Cu/l) resulted in 60, 106, and 90% increases on tolerance to Cu toxicity. These levels reduced growth but 0.18 of the incipient lethal level enhanced growth. Copper tolerance was lost after 3 weeks in control water. Four-week exposure to 0.59 of the incipient lethal level of Cu decreased Zn tolerance 56%. Acclimation of rainbow trout to sublethal levels of Cu, As, and HCN resulted in significant changes in tolerance of the toxicants by the fish.¹⁸⁷ Tolerance to As increased 47% after a 3-week acclimation period and tolerance to HCN decreased 32 and 15% after a 1- and 2-week acclimation period. Tolerance of Cu was dependent on the acclimation level of Cu. In a similar study,¹⁸⁸ the tolerance of rainbow trout to lethal levels of As and CN was measured following pre-exposure to sublethal levels of the same toxicants. Fish gradually increased their tolerances to As during a 3-week pre-exposure to 0.22 of the incipient lethal level whereas they decreased their tolerance to CN after 1 and 2 weeks of pre-exposure to 0.35 of the incipient lethal level. Neither As nor CN affected growth at these levels of pre-exposure. Genetic selection for Zn tolerance was studied by Rahel¹⁸⁹ in laboratory tests with flagfish (*Jordanella floridae*) and a natural population of shiners (*Notropis cornutus*). Increased Zn tolerance was observed after one generation in the flagfish but did not change with subsequent generations. Shiners in polluted streams were more tolerant to Zn than those in nearby unpolluted streams. One hypothesis proposed was that although fish may potentially evolve to withstand polluted conditions this process was too slow to allow them to survive contaminated conditions. Khangarot¹⁹⁰ studied the effects of Zn and Ni on four species of fish, *Puntius sophore*, *Rasbora daniconius neilgeriensis*, *Channa punctatus*, and *Lebistes reticulatus*. *Channa punctatus* were more sensitive to Zn and *Puntius sophore* were more sensitive to Ni. The 96-hour LC50 values for Zn ranged from 29.9 to 55 mg/l and 13.6 to 48.8 mg/l for Ni. Two laboratory bioassays were performed by Markarian *et al.*¹⁹¹ to determine the toxicity of metal mixtures and pH on the white sucker. Tests with mixtures of Ni, Cu, Zn, and Al and Ni, Cu, and Zn showed that Al mitigated toxicity of the other metals and that low pH increased toxicity. Muramoto¹⁹² exposed carp to aluminum chloride, aluminum sulfate, and mixtures of these compounds with NTA or EDTA. Addition of complexans decreased mortality and Al accumulation in both Al solutions. Kaur and Virk¹⁹³ found that hatchability of carp eggs decreased after exposure to concentrations of CuSO₄ greater than 0.1 mg/l. The decrease in hatchability was because of larval deformations and poor development in the post yolk-sac region. Concentrations of 3.5 to 4.0 mg CuSO₄/l resulted in 50% abnormal hatchlings. Eggs exposed to 5 mg CuSO₄/l died in cleavage stages. Static bioassays at 25°C using *Rasbora daniconius neilgeriensis* were performed by Durve *et al.*¹⁹⁴ with Cu. The LC50 values ranged from 2.1 mg/l for 12-hour tests to 0.2 mg/l for 96-hour tests. Khangarot¹⁹⁵ demonstrated that EDTA reduced Cu toxicity to guppies in 96-hour static bioassays. The effects of supplementary Cu in the diets of channel catfish were studied by Murai *et al.*¹⁹⁶ No signs of deficiency occurred in fish fed less

than 1.5 mg Cu/kg. Concentrations of 16 to 32 mg Cu/kg caused reduced growth, and some adverse effects occurred at levels of Cu greater than 32 mg/kg.

Dave *et al.*¹⁹⁷ studied the toxicity of Cd and eight solvent extraction chemicals on zebrafish, rainbow trout and *Daphnia magna*. In static tests, the 96-hour LC50 value for Cd and zebrafish was 1.7 mg/l. The LC50 values for the other chemicals are listed in Table 1. Yearling Atlantic salmon were exposed to concentrations of 1 to 1 000 µg Cd/l for 4.5 months.¹⁹⁸ At 1 mg Cd/l, the mean survival time was 3 days. Mixing humus in the water extended the survival time. The reduction in Cd toxicity was somewhat dependent on humus concentrations. Muramoto¹⁹⁹ studied the influence of EDTA and DTPA on Cd toxicity in carp exposed to contaminated water for 100 days. Both complexans decreased the toxicity of Cd to carp. Comparisons were made of Cd content in various parts of the fish in relation to the presence of both EDTA and DTPA. EDTA treatment of carp exposed to Cd also resulted in elimination of Cd from contaminated fish.²⁰⁰ In another study, the effects of Cd on carp were analyzed by Muramoto.²⁰¹ Fish with deformed vertebrae appeared on Days 47, 85, and 73 of experiments with 0.01, 0.05, and 0.1 mg Cd/l, respectively, and on Days 58 and 95 after exposure to 0.01 and 0.5 mg Cd/l in a second experiment. The frequency of development of deformed fish was not constant and deformed fish did survive the experiment. Chromium toxicity and gill accumulation in goldfish was studied by Riva *et al.*²⁰² Chromium toxicity increased with dose and temperature. There was a slight increase in Cr⁶⁺ concentration in the exposed fish and the presence of the fluorescent whitener (Blankophore) did not significantly alter Cr⁶⁺ toxicity. A study on the toxicity, accumulation and depuration of tetramethyllead (Me₄Pb)²⁰³ showed that juvenile rainbow trout were sensitive to this compound at 51 µg/l. Trout accumulated Me₄Pb rapidly with the highest concentrations found in the intestine. Depuration was rapid at first, then decreased slowly.

The accumulation of Cd, Cr, and Pb by bluegill sunfish under O₂ and temperature stress was studied by Freeman.²⁰⁴ Fish were exposed for 7 days to 0.5 µg/ml of each toxicant. Accumulation of Pb in gill tissue was found to be related to DO while temperature greatly influenced accumulation of all three metals in all other tissues. The uptake of Cd in perfused gills of rainbow trout was investigated by Part and Svanberg.²⁰⁵ Cadmium was transferred through perfused gills and addition of EDTA decreased Cd transfer through the gills. Vertebral deformities in carp caused by a 73-day exposure to Cd were investigated in another study.²⁰⁶ Calcium concentrations in gills, viscera, and vertebrae were lower in treated fish than control fish. Phosphorus concentrations in the vertebrae also decreased through Cd exposure. Michibata²⁰⁷ investigated the dependence of Cd accumulation in the egg chorion on water hardness and the correlation between adsorbed Cd and egg survival in *Oryzias latipes*. Eggs raised in a medium with a high water hardness were the most resistant to Cd. Accumulation of Cu in carp gills was studied by Drbal and Svobodova.²⁰⁸ Accumulation of Cu in gills of fish exposed in well water was 3.5 times higher than the controls and 1.8 times higher than the controls in fish exposed using pond water. Marathe and Deshmukh²⁰⁹ studied the effects of Cu in the aquatic food chain. *Channa gachua* were fed guppies that were

exposed to 100 $\mu\text{g Cu/l}$ for 48 hours. Fish fed contaminated guppies showed a 94% weight loss in 30 days while fish fed uncontaminated guppies had a 157% weight gain.

The disappearance of Hg and Se from aquarium water and their uptake by goldfish were observed by Sharma and Davis.²¹⁰ The average concentration factor for methylmercuric chloride was 10 times greater than for mercuric chloride while selenomethionine was concentrated 40 times faster than sodium selenite. The authors concluded that the BCF of a toxicant may be more important in determining its level in fish than the initial concentration in water. Modification of the toxicity of mercuric chloride and methylmercuric chloride by sodium selenite and L-selenomethionine, and the interaction of Se on the accumulation of Hg from water by goldfish was also observed by Sharma and Davis.²¹¹ Selenium was not found to protect against Hg poisoning. The authors found greater Hg accumulation in surviving fish than dead ones while the reverse was true for Se. Edible muscle of several fish including largemouth bass, smallmouth bass (*Micropterus dolomieu*), bullhead, muskellunge (*Esox masquinongy*), perch, northern pike, and walleye (*Stizostedion vitreum vitreum*) were analyzed for Hg and Se content and chemical form.²¹² The Se and Hg content in fish were not correlated. Sixty to 95% of the total Hg present was methylmercury and 15 to 35% of the total Se present was selenate for most species. Petukhov and Storozhuk²¹³ studied the effects of Hg on mortality and tissue accumulation in larval chum (*Oncorhynchus keta*) and larval coho. It was concluded that the toxic effect of Hg was the result of Hg blocking the biosynthesis of proteins and replacing biologically active metals in metalloproteins. The accumulation of Hg in the tissues of rainbow trout was measured by Ribeyre and Boudou.²¹⁴ The concentration of Hg in brain, muscles, posterior intestine, gills, and liver was dependent on the length of exposure and tissue type. In another study,²¹⁵ rainbow trout were exposed to 1 $\mu\text{g Hg/l}$ at 10, 18, and 26°C. Results indicated that Hg was transferred and accumulated in different organs through direct absorption. Tests on the effects of temperature on transfer rates for methylmercury between trophic levels²¹⁶ showed that at 18°C the rate of transfer of Hg from the water to the terminal consumer in a four-level trophic chain was 15%. After 30 days, rainbow trout exposed to 1 mg Hg/l contained 700 $\mu\text{g/organism}$ and elevated temperatures reduced the transfer rate. Carp were exposed to Hg for up to 180 days.²¹⁷ Tissue analysis showed that enrichment factors for Hg varied with length of exposure and to levels of Hg in the water prior to exposure. Mondain and Gras²¹⁸ studied the accumulation of Hg and methylmercury in goldfish. Inorganic Hg was accumulated less than methylmercury. The transfer factors were 90 and 760 to 780, respectively. The bioaccumulation of methylmercury in guppies was examined by Stary *et al.*²¹⁹ Methylmercury absorbed on fish food remained completely in fish and was released with the biological half-life of 110 days. The accumulation of methylmercury from water was found to be very rapid and the accumulation constant was 237/d.

In a 31-day study of the common eel (*Anguilla anguilla*), Bell *et al.*²²⁰ found that the rate uptake of ⁴⁸V-orthovanadate was proportional to environmental concentrations. In an 8-week test at 10^{-8} M V, the rate of uptake was 1.8 pmol/h · 100g body weight. Death occurred in eels exposed to 10^{-4} M

orthovanadate for 7 to 26 days. Uptake of V by the eel was found to be the greatest in liver tissue.²²¹ Variations in the amount of V in the kidney, bone, carcass, and liver were not found to be significant over a 5-week depletion period. Five-day-old guppies were exposed to Zn for 134 days in bioassays performed by Pierson.²²² Whole-body Zn concentrations decreased with growth but increased with the onset of pregnancy. Zinc was transferred from the female parent to the embryos. After 134 days, 0.6 mg Zn/l caused reduced weight and a delay to and decrease in the first brood. Zinc concentrations of 0.2 mg/l delayed sexual maturity after a 70-day exposure. The relationship between dietary Zn levels and Zn concentration in the common carp was evaluated by Jeng and Sun.²²³ Zinc was accumulated in fish fed diets containing Zn concentrations greater than 1 007 $\mu\text{g/g}$. Dietary Zn levels < 294 $\mu\text{g/g}$ did not result in significant Zn accumulation in the tissues. The As content of several tissues of the muscle of the monkfish (*Kathetostoma giganteum*), English hake (*Merluccius australis*), and Hoki (*Macruronus novaezelandiae*) were measured by Kobayashi *et al.*²²⁴ In the monkfish, As levels were higher (5.6 to 25.4 $\mu\text{g/g}$) in the muscle than in other tissues, the Hoki had higher As levels in other tissues than in the muscle, and the hake showed little difference between other tissue and muscle. Hilton *et al.*²²⁵ measured the dietary Se requirements of rainbow trout and studied responses to excessive levels of dietary Se. Chronic dietary Se toxicity occurred at 13 $\mu\text{g Se/g}$ dry feed. Selenium uptake and tissue accumulation were potentially toxic to trout reared on diets in excess of 3 $\mu\text{g/g}$. Maximal plasma glutathione peroxidase activity was found at 0.15 to 0.38 $\mu\text{g/g}$ dry feed. This level was less than the average Se concentration in many commercial diets.

The sublethal effects of nitrite on channel catfish were examined by Huey *et al.*²²⁶ A 24-hour exposure to 1 to 5 mg/l of nitrite increased methemoglobin concentrations in the blood. The transfer of fish to fresh water following a 6-hour exposure resulted in near normal amounts of methemoglobin after 24 hours. An inverse relationship was noted between pH and methemoglobin formation after exposure to nitrite. Several salts suppressed methemoglobin formation except for sodium sulfate which had no effect. In a series of 96-hour bioassays using rainbow trout, Russo *et al.*²²⁷ found nitrite toxicity to be pH-dependent within the range considered desirable for aquatic life (pH 6.5 to 9.0). Both NO_2^- and HNO_2 were found to be toxic to the fish where it was previously thought only the free acid (HNO_2) was toxic. The presence of chloride, sulfate, phosphate, and nitrate were also found to influence nitrite toxicity. Blanco and Meade²²⁸ fed ascorbic acid at four levels to steelhead trout (*Salmo gairdneri*) and exposed them to concentrations of 5, 10, and 15 mg nitrite/l. When levels of ascorbic acid were higher (2 000 mg/kg), the tolerance to nitrites increased, however, the increase was less in large trout (37 to 42 g) than in small trout (3.2 to 3.5 g) fed identical amounts of ascorbic acid. When the temperature was raised, the percent methemoglobin in their blood increased. Juvenile channel catfish were exposed to nitrite for 31 days by Colt *et al.*²²⁹ Nitrite concentrations greater than 1.6 mg/l caused reductions in growth and a nitrite concentration of 3.7 mg/l significantly increased mortality. Bath and Eddy²³⁰ measured nitrite transport across the gills of rainbow trout. Exposure

to water containing 0.7 mM/l of NO_2^- for 24 hours resulted in blood plasma levels of 5 mM NO_2^- /l, which demonstrated the ability of the fish to transport NO_2^- against a concentration gradient and bioaccumulate the chemical in the blood plasma.

Cairns *et al.*²³¹ exposed bluegills to sublethal concentrations of heavy metals and monitored their gill ventilation rates. The response to toxic solutions of Zn, Cu, Ni, and Cr was an increase in the ventilatory rate as well as a decrease in signal amplitude. Fish were found to be capable of reacting to pulses of these toxicants in a similar manner. The effect of sodium arsenate on algae, *Daphnia*, and mosquitofish were observed by Jurewicz and Buikema.²³² At LC50 concentrations of 49 to 57 mg/l, loss of equilibrium, lack of motility, and hemorrhaging of the gill and opercular region were observed. Copper pharmacokinetics were determined in the gills of the pumpkinseed sunfish (*Lepomis gibbosus*) by Anderson and Spear.²³³ Juvenile and adult sunfish accumulated Cu in their gills. Cu was eliminated in fish transferred to clean water. Accumulation was found to be biphasic with an initial fast phase followed by a slow phase and elimination was monophasic. The biological half-life was 1.6 hours for a 3.5-g sunfish. Kumar and Pant²³⁴ recorded histopathologic effects of acutely toxic levels of CuSO_4 and ZnSO_4 on *Puntius conchonius*. Severe gill damage and necrotic changes in the liver and kidney resulted from 0.6 and 34 mg/l of these compounds, respectively, after 48 hours. Effects on the kidneys and liver were more severe than on the gills. Also, while Zn was less toxic than Cu, the damage it caused in the kidney was more severe. Thomson²³⁵ investigated the possible association of Cu pollution with vibriosis in the eel. Forty-two- to 64-day tests with eels in fresh water and brackish water indicated that Cu was not the initiating factor in developing bacterial infection. The susceptibility of steelhead trout to redmouth infection following Cu exposure was illustrated by Knittel.²³⁶ Copper, at concentrations of 7 and 10 $\mu\text{g/l}$ for 96 hours, caused more fish to die of infection than in the controls. Infection susceptibility increased with time of exposure to a single dose (10 $\mu\text{g/l}$), reaching a maximum after 48 hours. However, a Cu concentration of 10 $\mu\text{g/l}$ seemed to inhibit infection compared to lower Cu concentrations. Exposures of brown trout and carp to four heavy metals for 38 weeks were conducted by O'Neill²³⁷ to examine their effect on the fishes immune responses. Concentrations of 0.75 mgNi/dm³, 1.06 mgZn/dm³, 0.29 mgCu/dm³, and 1.01 mgCr/dm³ suppressed humoral antibody responses.

Sastry and Gupta²³⁸ determined the 96-hour LC50 of lead nitrate in *Ophiocephalus punctatus* and *Heteropneustes fossilis* to be 13.2 mg/l and 12.0 mg/l, respectively. Sublethal concentrations (3.0 and 2.8 mg/l) did not affect succinic, lactic, and pyruvic dehydrogenases in short-term exposures but chronic exposures (greater than 15 days) resulted in the inhibition of enzyme activity. Changes in basophilic stippled erythrocyte counts and in δ -amino-levulinic dehydratase activity were studied in adult carp and in adult European dogfish (*Scyliochinus canicula*) exposed to Pb.²³⁹ Maximal averages stippled cell count was obtained in carp at sublethal concentrations after 38 days of exposure. Williams and Wootton²⁴⁰ studied the effects of CuSO_4 (0.5 mg/l) and formalin (200 mg/l) on the blood chemistry of rainbow trout. Exposure of Cu for 1 hour elevated the mean values of all parameters measured. Formalin only significantly affected hematocrit and GOT activity. Recovery after a single treatment and degen-

erative changes resulting from repeated treatments were described. An air-breathing fish (*Heteropneustes fossilis*) and a non-air-breathing fish (*Labeo rohita*) were exposed to 40 mg Cd/l for 2 weeks to compare the effects of Cd on carbohydrate metabolism and other metabolic activities.²⁴¹ Although both fish showed stress from Cd exposure metabolic manifestations of stress were quite different. Agrawal and Srivastava²⁴² measured the effects of Mn on blood dyscrasia in adult female *Colisa fasciatus*. Significant decreases in total erythrocyte count and the hepatosomatic index were found after a 90-hour exposure to 2 500 mg MnSO_4 /l. The 96-hour LC50 was 2 850 mg/l.

Rainbow trout were exposed by Lock *et al.*²⁴³ to mercuric chloride (10 to 2 000 $\mu\text{g/l}$) and methyl mercuric chloride (5 to 50 $\mu\text{g/l}$) for 4 hours, 24 hours, and 1 week. Lethal concentrations of both compounds inhibited Na^+ , K^+ , and ATPase activity in the gills. Sublethal concentrations decreased Na^+ and Cl^- plasma concentrations and increased water uptake by the gills. Rainbow trout exposed to higher concentrations of HgCl_2 (50 to 2 000 $\mu\text{g Hg/l}$) and methyl mercuric chloride (5 to 100 $\mu\text{g Hg/l}$) for the same periods of time²⁴⁴ contained increased mucous secretion at all exposures. Mercuric chloride elicited a stronger response than methyl mercuric chloride at the same concentration. Gupta and Sastry²⁴⁵ studied the effects of HgCl_2 on the activities of succinic, lactic, and pyruvic dehydrogenases in the digestive systems of *Ophiocephalus punctatus* and *Heteropneustes fossilis*. Maximum inhibition occurred after 15 days of exposure to 0.3 mg/l. *Heteropneustes fossilis* was the more sensitive of the two species studied. Nine species of freshwater teleosts were exposed to mercuric nitrate (5 to 20 mg Hg/l) for 4.5 hours in tests by Shaffi.²⁴⁶ Liver and muscle glycogen levels increased at 5 mg/l. Renal glycogen decreased with increasing Hg concentrations. Brain glycogen increased at concentrations less than 10 mg/l but decreased at higher levels. Glucose and lactate levels increased with increasing Hg levels. *In vitro* effects of Zn^{2+} on branchial ATPases from rainbow trout were studied by Watson and Beamish.²⁴⁷ Inhibition of all the enzymes studied occurred at 1 to 100 mg Zn^{2+} /l in tests at 11°C. Hydrolysis of ATP resulted from incubation with Zn^{2+} perhaps because of cation replacement in cofactors or substrates. Maximum specific activity of this hydrolysis occurred at 150 mg Zn^{2+} /l. Khan-garoi²⁴⁸ exposed *Channa punctatus* to 56 mg Zn/l for 8 and 24 hours and both exposures resulted in increased ATPase activity in the gills. The effect of 3-hour exposure to 20 to 25 mg Zn/l on tissue glycogen content in nine fish was studied by Shaffi.²⁴⁹ The results indicated that tissues responded differently to the Zn levels, however, the carps were less resistant than the other fishes studied. Chitra and Rao²⁵⁰ studied the effects of NaF on blood and liver enzymes in fish (*Channa punctatus*). An LC50 concentration of 10 mg/l caused a significant increase in blood cholesterol, glucose, and AChE at 30°C but decreased blood glucose and O_2 consumption at 15°C. Enzymatic changes were noted in liver extracts at both temperatures. The physiological significance of the above changes were discussed.

Organics. Welch *et al.*²⁵¹ provided quantitative data on the background hydrocarbon residues in fishes from the Great Lakes and eastern Montana using GC/MS procedures. Residues of n-heptadecane in lake trout from four Great Lakes ranged from 21 to 240 $\mu\text{g/g}$ in the lipids. Data on Montana

fish samples showed that the total n-hydrocarbon residues ranged from 1.3 to 160 $\mu\text{g/g}$ for several species of fish. Fish composite samples collected in 1978 near river mouths or confluences of tributaries in the Great Lakes watershed were analyzed for PCBs and other related chemicals by Veith *et al.*²⁵² Total PCBs in whole fish composites ranged from 0.13 to 14.6 $\mu\text{g/g}$ and 65% of the samples contained $>2 \mu\text{g/g}$. DDT and all its metabolites were identified in all samples. Hexachlorobenzene was found in 65% of samples and chlordane was found in 38%. Petroleum hydrocarbons and chlorobenzenes, chlorostyrenes, chlorophenols, and chlorinated aliphatic compounds were also identified. Sloterdijk²⁵³ reported the concentrations of PCB, Hg, and five other metals found in the lateral muscle of the northern pike from the province of Quebec. Mercury levels were high and ranged from 0.1 to 5 $\mu\text{g/g}$ and PCBs ranged from non-detectable levels to 12 $\mu\text{g/g}$. Organochlorine residues in fish from Lake Mjøsa in Norway were examined by Brevik.²⁵⁴ The data showed that average levels (fat weight basis) of PCBs, HCB, and sum-DDT for the sampling years of 1974 to 1979 decreased but changes were not found to be significant. Dougherty *et al.*²⁵⁵ examined the presence of xenobiotic chemicals in fish and humans using negative chemical ionization studies. Polychloronaphthalenes, polychlorostyrenes, polychlorobiphenyls, polychlorodibenzofurans, and polychlorodibenzodioxins, as well as other chemicals were found in fish from rivers in the midwestern and northeastern U. S. Only fish from the Tittabawassee River, Michigan, contained polychlorodibenzo-dioxins. The polychlorodibenzofurans appeared in fish samples significantly contaminated ($>2 \mu\text{g/g}$) with polychlorobiphenyls. Composite fish samples collected from the Ashtabula River, Ohio, and Wabash River, Indiana, resulted in the identification of a series of chlorinated alkanes, chlorinated alkenes, chlorinated alkylamines containing two of five carbons and polychlorinated styrenes.²⁵⁶ Fish samples from Wabash River contained a series of chlorinated norbornenes, pentachlorobenzyl alcohol, and pentachlorophenol (PCP). High levels of 4,4'-methylene bis(2-chloroaniline) and 2-chloroaniline (2CA) were measured in the water and the sediment of an industrial waste lagoon and in the surface runoff to the south branch of the Raisin River at Adrian, Michigan.²⁵⁷ A concentration of 13 $\mu\text{g/kg}$ of 2CA was detected in edible tissues of two white suckers found near a plant outfall. The authors suggested that 2CA was bound to macromolecules rather than free in tissues. Yamagishi *et al.*²⁵⁸ identified musk xylene (5-tert-butyl-2,4,6-trinitroxylenes) and musk ketone (2-acetyl-5-tert-butyl-4,6-dinitroxylenes), artificial perfumes in goldfish collected from the Tama River, Tokyo. The levels of musk xylene and musk ketone were 0.2 and 0.05 $\mu\text{g/g}$, respectively. In the same samples, the residue levels of dieldrin and p,p'-DDE were 0.001 and 0.015 $\mu\text{g/g}$, respectively.

The toxic effects and mutagenic capacity of water from the Sava River (Yugoslavia) on carp were assessed by Kurelec *et al.*²⁵⁹ Fish living in the investigated segment of the river were highly induced with respect to their benzo(a)pyrene monooxygenase (BPMO) activity. The intraperitoneal injection of hexane extracts of Sava River water failed to induce liver BPMO. No correlation to low water quality was observed with the frequency of neoplasms in wild fish. A multigeneration fish breeding study was conducted by Newsome²⁶⁰ to determine the effects of trisodium carboxymethylsuccinate (NaCMOS) on all life stages and fecundity on the convict cichlid

(*Cichlasoma nigrofasciatum*). No effect on fecundity, survival of eggs and fry, or adult breeding behavior was found at 100 mg NaCMOS/l. The author noted that the 100 mg/l concentration of NaCMOs was two orders of magnitude greater than the predicted aquatic environmental concentration under the most unfavorable conditions of its use, wastewater treatment, and discharge.

The 96-hour static acute toxicity of 64 priority pollutants to the bluegill was determined by Buccafusco *et al.*²⁶¹ The chemicals tested consisted of 10 benzenes, 7 chlorinated ethanes, 3 ethers, 14 phenols, 4 dichloropropanes/propenes, 5 phthalate esters, 4 ethylenes, and 17 other compounds (Table 1). Curtis and Ward²⁶² determined the aquatic toxicity of 40 industrial chemicals (mostly organic) utilizing the fathead minnow (Table 1). Ten chemicals were tested with the grass shrimp (*Palaemonetes pugio*). Mercuric thiocyanate was the most toxic compound with a 96-hour LC50 value of 0.15 mg/l in freshwater. Several organic compounds were not toxic at levels up to 600 mg/l. The quantitative structure-activity relationships in toxicity studies with guppies and 50 organic industrial chemicals were studied by Könnemann.²⁶³ Materials with a $\log P < 6$ showed a good correlation between toxicity and $\log P$ while those with a $\log P > 6$ did not. The acute toxicity of 1,1-dichloroethylene to fathead minnows and *Daphnia magna* was observed by Dill *et al.*²⁶⁴ (Table 1). Using static test conditions *D. magna* were considerably more sensitive (48-hour LC50 = 11.6 mg/l) than fathead minnow (96-hour LC50 = 169 mg/l). The toxicity of chloroform, bromodichloromethane, dibromochloromethane, and bromoform to carp embryos was investigated by Mattice *et al.*²⁶⁵ The LC50 values corrected to time-weighted mean concentrations resulted in weighted LC50 values of 97.2 mg/l for chloroform, 67.4 mg/l for bromodichloromethane, 33.5 mg/l for dibromochloromethane, and 52.3 mg/l for bromoform. In addition, the period of water-hardening of fertilized eggs was not critical for expression of toxicity of dibromochloromethane. The acute toxicity of phenol, PCP, and dinitrophenol (DNP) to patra (*Notopterus notopterus*), singii (*Saccobranchius fossilis*), and kharda (*Colisa fasciatus*) was measured by Verma *et al.*²⁶⁶ Patra were the most sensitive to all three compounds (Table 1). In other tests,²⁶⁷ the toxicity of phenol, PCP, and DNP were tested individually and in combination to that of patra. The 96-hour LC50 values for the single chemicals are given in Table 1. The combinations of phenol + DNP/PCP was the most synergistic, PCP + DNP/phenol was antagonistic, and DNP + phenol/PCP was additive in its effects. Dalela *et al.*²⁶⁸ exposed three species of fish to phenol, DNP, and PCP at four pH levels to evaluate the toxicity at each pH level (Table 1). A change in pH from 7.3 to 8.8 decreased toxicity. The acute toxicity of phenol and 11 substituted phenols to the fathead minnow was studied by Phipps *et al.*²⁶⁹ (Table 1). The most toxic compound was PCP with a 96-hour LC50 value of 0.22 mg/l, while the least toxic was 3-methoxyphenol with a 96-hour LC50 value of 76 mg/l. McLeese *et al.*²⁷⁰ assessed the lethality and bioconcentration of alkylphenols to shrimp and to juvenile Atlantic salmon. The LC50 values were found to decrease with increasing octanol/water partition coefficient. The BCF for salmon increased with increasing octanol/water partition coefficient and ranged from 37 for butylphenol to 6000 for dodecylphenol. Adema and Vink²⁷¹ compared the toxicity of PCP, 1,1,2-trichloroethane, dieldrin, and 3,4-di-

chloroaniline to the guppy. Values for 48-hour to 60-day tests are listed in Table 1. The biodegradation and short-term toxicity of eight amines to algae (*Selenastrum capricornutum*), *Daphnia magna* and rainbow trout were determined by Calamari *et al.*²⁷² The toxic effects of the eight amines were quite different for each species. The minimum and maximum 96-hour LC50 for rainbow trout were 37 to 380 mg/l in hard water and 5.5 to 180 mg/l in soft water. The rank order of toxicity was also very dissimilar for all species.

Two freshwater teleosts were exposed to one nonionic and four anionic synthetic detergents under static conditions for 96 hours by Verma *et al.*²⁷³ (Table 1). The relative potency of the detergents was in decreasing order from the nonionic to anionic for both fishes, respectively, and the relative susceptibility was highest for nonionic than ionic for *Channa punctatus* compared to *Saccobranchius fossilis*. Dalela *et al.*²⁷⁴ exposed *Saccobranchius fossilis* to sodium lauryl sulfate (SLS) for 30 and 60 days on a static renewal basis. Coagulation time of the blood was reduced at 2.24 mg/l at 30 days and at 1.12 mg/l at 60 days. Significant increases in hematocrit were noted at 2.24 mg/l for both exposure periods. Pittinger and Tennant²⁷⁵ investigated the acute lethal toxicity of SLS⁸ to the fathead minnow and the critical surface tension below which no mortality was seen. The effects of water hardness on critical surface tension levels and corresponding toxicity was also studied.

Rainbow trout eggs, yolk-sac fry, and juveniles were exposed to 2,3,7,8-tetrachlorobenzo-p-dioxin (TCDD) for 96 hours and were then held in untreated water to determine what effects TCDD had on development.²⁷⁶ Significant growth retardation was seen at 72 days post-exposure at 0.1 ng/l. Higher concentrations of 1 and 10 ng/l resulted in significant numbers of developing edemas and death. Various histological effects were also seen for the yolk-sac fry and juvenile. Moles *et al.*²⁷⁷ observed significantly reduced growth in coho salmon fry exposed for 40 days to sublethal concentrations of naphthalene and toluene (1.4 mg/l and 5.8 µl/l, respectively). Fry exposed to naphthalene had a slower growth rate than fry exposed to equivalent (percentage of 96-hour LC50) concentrations of toluene. Concentrations 18% of the LC50 of naphthalene and 26% of the LC50 of toluene had no effect on weight or length of fry. The growth of cutthroat trout exposed at four concentrations (ranges 100 to 520 µg/l) of Wyoming crude oil for 90 days was significantly slower than in control fish.²⁷⁸ Survival at 520 µg/l was 52% and concentrations of 450 and 520 µg/l induced gill lesions and development of lesions of the retina and lens of the eye of the trout. The accumulation of total hydrocarbons in fish tissue was found to be directly related to water concentration, except at 52 µg/l. Alkylated mono and dicyclic aromatic hydrocarbons were accumulated most readily and the dominant aromatic components in oil, water, and fish were naphthalenes.

Southworth *et al.*²⁷⁹ compared the observed and predicted bioconcentration of azaarenes in the fathead minnow. The bioconcentration in fish of those azaarenes with moderate or high (>1 000-fold) predicted BCFs was found to be lower than predicted or observed in *Daphnia pulex*. The deviation was attributed to the metabolic alterations of azaarenes in the fish. Benz(a)pyrene levels in the muscular tissue and various organs of carp, brown trout, seatrout, and rainbow trout were reported by Veldre *et al.*²⁸⁰ The amount of benz(a)pyrene in various

organs was very similar to the muscular tissue and the average level of benz(a)pyrene in fish was <1 mg/kg. Wong *et al.*²⁸¹ found ¹⁴C-diphenyl ether to be taken up rapidly by both rainbow trout in freshwater and sticklebacks (*Gasterosteus aculeatus*) in seawater. The amount of radioactivity accumulation increased with length of exposure but the radioactivity levels decreased biphasically with time (35 and 55%, respectively) when fish were placed in clean water. The rate of diphenyl hydroxylation by speckled trout (*Salvelinus fontinalis*) liver microsomes was determined to be low, 288 nmol/g liver · h. The fate and effects of several hydrocarbons and chlorinated hydrocarbons to rainbow trout, carp, and sunfish (*Lepomis macrochirus*) were examined by Lech and Melancon.²⁸² ¹⁴C-Naphthalene, 2-methylnaphthalene, 1,2,4-trichlorobenzene, PCP, and pentachloroanisole were taken up rapidly by the trout. Increased duration of exposure to ¹⁴C-naphthalene or ¹⁴C-2-methylnaphthalene affected the elimination of the ¹⁴C containing components in the fish. Interactions between the chemicals and the fish were also evaluated by assessing the effects of inducing agents on hepatic metabolizing enzymes (*in vitro*) and on the effects of inducing agents on the metabolism and the deposition of the other chemicals *in vitro*. Nava and Engelhardt²⁸³ evaluated the dynamics of uptake and clearance, as well as tissue distribution, of ingested ¹⁴C-naphthalene concentration of 10 and 100 µl/kg in the American eel (*Anguilla rostrata*). Similar patterns were seen in the analysis for the uptake and clearance in both the solvent extractable and the water soluble fractions. At all times, radioactivity levels were significantly higher in the bile than in other tissues; approximately 45 times higher levels of maximum uptake was observed in the bile than those observed for the liver which exhibited the highest tracer activity levels. A 10-fold greater content of hydrocarbons was detected in the bile and tissues at 100 µl/kg as compared to 10 µl/kg. The uptake, elimination, and metabolism of phenol, 2,4,5-trichlorophenol, and p-nitrophenol by fathead minnows exposed to sublethal concentrations of radiolabelled compounds was measured by Call *et al.*²⁸⁴ Uptake and concentration in the fish was greatest with phenol. 2,4,5-trichlorophenol was eliminated from fish at a faster rate than phenol or p-nitrophenol while the ¹⁴C depuration half-life was the longest with phenol.

Califano *et al.*²⁸⁵ studied the uptake, retention, and elimination of PCB (Aroclor 1254) by larval striped bass (*Morone saxatilis*). The bass removed PCB from Hudson River water rapidly and nearly completely. Maximum uptake was 48 hours after exposure (60% available PCB taken up) and more than 80% of uptake was after 12-hour exposure. The bass eliminated 1% of PCB in first 24 hours and 18% after 48 hours. Hora²⁸⁶ found the removal of the skin from carp filets significantly reduced the PCB concentrations and the lipid content of the sample. The overall reduction of PCBs in the filet without skin compared to filets with skin was 26%, and overall lipid reduction was 30%. Skinned filets from the largest weight class of carp (>23 kg) resulted in the least effect on PCB and lipid removal. Fingerling lake trout was exposed to various combinations of DDT, DDE (100 ng/l), and PCBs (100 and 500 ng/l) for 12 weeks and PCBs alone for an additional 16 weeks to test the hypothesis that PCB levels accumulated in fish may have influenced the uptake and elimination of DDE and DDT in fish from Lake Michigan.²⁸⁷ PCBs were not found to influence the accumulation or elimination rates of DDE or

DDT in fish. Yellow perch (*Perca flavescens*) and rainbow trout exposed to 2,5,2',5'-tetrachloro-[^{14}C]biphenyl (4-CB) via oral or intraperitoneal routes eliminated 20 to 30% of the radioactive compound 3 to 4 days after treatment, but very little after that.²⁸⁸ The viscera and carcass were the main distribution sites for 4-CB and skeletal muscle, skin, and scales were the minor sites. However, in various rainbow trout, the major distribution sites were the skeletal muscle and carcass; the viscera and skin were the minor sites.

Replicate 28-day exposures with 1,2,4-trichlorobenzene (TCB), hexachlorobenzene (HCB), and p,p'-DDE (DDE), and fathead minnow were run to evaluate the ASTM method for measuring BCFs. The mean BCFs were 1 700 for TCB, 35 000 for HCB, and 50 000 for DDE.²⁸⁹ The results showed that steady-state residues were not attained for highly bioaccumulative chemicals after 28 days and calculation of the BCF by dividing the 28-day residues by the mean water concentration was inadequate. Smith *et al.*²⁹⁰ studied the assimilation efficiency and clearance of TCBs in rainbow trout. Food containing TCBs also incorporated a radioactive chemical marker. The trout assimilated approximately 61% of the TCBs when given a pulse dose meal that contained 50 μg TCB/g. Individual fish cleared more than 90% of TCBs in tissues 174 hours after dosing. Rainbow trout were found to take up ^{14}C -ring-labelled triphenyl phosphate at lower rates in water from the Red River, Canada, than in dechlorinated city water.²⁹¹ The liver accumulated the highest residues and experiments to determine the uptake and clearance rates resulted in a bioaccumulation ratio of 2 950. Tritium-labelled dodecylcyclohexane was given *per os* to rainbow trout, and after 7 days the percentage of ^3H was distributed as 20% in feces, 19% in urine, 30% in test water, and 26% in whole carcass.²⁹² The elimination was at the maximum level 18 to 24 hours after dosage and more than 15% of ^3H ingested was excreted in urine in 4 days. Seventy-seven percent of the radioactivity of the carcass was from the ^3H -dodecylcyclohexane and the highest level of radioactivity was found in the adipose tissue. The authors estimated an absorption level of 75% of the ingested hydrocarbon.

Kimerle *et al.*²⁹³ exposed bluegills for 30 days to ^{14}C -linear alkylbenzene sulfonate (LAS) at 0.5 mg/l. When all radioactivity was assumed to be intact LAS, the whole-body BCF was estimated to be 104. The BCF in muscle was 36 and 5 000 in the gall bladder. The liver, gills, viscera, blood, and remaining carcass had BCF values which ranged from 64 to 283. The rapid clearance of radioactivity in all organs and tissues was observed with half-lives of 2 to 5 days.

The eastern mudminnow (*Umbra pygmaea*) was exposed to two genotoxic agents, benzo(a)pyrene and ethyl methane-sulfonate, and Rhine River water.²⁹⁴ Both compounds induced chromosome aberrations in the eastern mudminnow under laboratory conditions. Two tests conducted in the Rhine River showed an effect on the frequency of both sister chromatid exchange and chromosome aberrations. Pfeifer *et al.*²⁹⁵ compared the carbon tetrachloride-induced hepatotoxic response in rainbow trout by measuring the alanine aminotransferase (GPT) activity in plasma from fish fed two commercial diets. At 3, 6, 12, and 18 hours after treatment with 1.0 mg CCl_4/l , the plasma enzyme activities for the two groups of fish fed these diets were significantly different. No correlation was found between the degree of hepatocellular damage and ele-

vated GPT activity or the diet. The reduction of chlorinated nitrobiphenyl ethers and their nitroso derivatives, and the stability of their amino derivatives by carp and rat intestinal microorganisms were investigated by Miyauchi *et al.*²⁹⁶ Under aerobic and anaerobic conditions, all chlorinated nitrobiphenyl ethers used were reduced to the corresponding amino derivatives by the intestinal microorganisms of the carp and rats. The reduction rate was greater in the rate under both conditions. Miyauchi and Takagi²⁹⁷ assessed the ability of the white-spotted char (*Salvelinus leucomaenis*) liver homogenate to convert chlorinated nitrobiphenyl ethers and their nitroso and amino derivatives to mutagens using the Salmonella/microsome test. p-Nitrobiphenyl ether, 3-chloro p-nitribiphenyl ether, 4-chloro p-nitrobiphenyl ether, and all nitroso derivatives induced mutations in *Salmonella typhimurium* without liver homogenate. Mutagenic activities of the above ethers with the liver homogenate increased while the nitroso derivatives greatly increased. Without the liver homogenate the amino derivatives showed no mutagenic activity but did with the liver homogenate.

Two fatty alcohols, hexadecanol and octadecanol, were tested for their effects on the respiratory functions of rainbow trout.²⁹⁸ Using confinement test conditions, a significant drop of the O_2 -dependence level occurred only in fish during exposure to 1 000 mg/l of octadecanol. Increases in O_2 consumption were noted during the recuperation period in pure water and 2 hours following exposure at 10, 100, and 1 000 mg/l of fatty alcohols. A behavioral study of the single, combined, and uptake effects of n-butanol and n-hexanol in goldfish was conducted by Hill *et al.*²⁹⁹ Concentrations of the alcohols were increased by six equal steps. After each step increase, the initial conditioned response was decreased and was related to the size of the step and the initial concentration. Recovery of the goldfish was observed at final concentrations (by step) of <15 mM n-butanol and 1.06 mM n-hexanol; while recovery was almost complete for lower concentrations, it was only partial at 12.5 and 15 mM butanol. The uptake of n-butanol in the brain was measured as a function of time following step changes and indicated an acute tolerance of goldfish to n-butanol and n-hexanol. A mixture of monocyclic aromatic hydrocarbons at 3 to 4 mg/l was avoided by pre-smolt coho salmon while smolting coho salmon avoided the mixture at concentrations of less than 2 mg/l.³⁰⁰ When the three components of the mixture, benzene, toluene, and o-xylene, were tested separately, the salmon avoided the chemicals at lower concentrations than the total mixture concentration; o-xylene was the most repellent at 0.2 mg/l. The chemosensory modality of the olfactory bulb was not disrupted at 4 mg/l and the olfactory system was stimulated by the mixture of aromatic hydrocarbon mixture at concentrations that correlated with the observed avoidance behavior. During the peak run of the salmon migration in Chambers Creek, Washington, mature Pacific salmon (*Oncorhynchus* sp.) were exposed to a mixture of monocyclic aromatic hydrocarbons by Weber *et al.*³⁰¹ The salmon substantially avoided the petroleum hydrocarbons at concentrations of 3.2 mg/l and higher.

Breger *et al.*³⁰² studied the metabolism of 2-methylnaphthalene to isomeric dihydriols using the hepatic microsomes of the rat and the rainbow trout. The conversion of 2-methylnaphthalene to both monohydroxylated compounds and dihydriols was reduced by incubation with carbon monoxide,

the omission of NADPH, or use of heat-denatured microsomes. The three dihydriols obtained *in vitro* in microsomes from both rat and fish were identical with compounds isolated from rat urine. Henderson *et al.*³⁰³ tested four concentrations of hydrazine on the mid-cleavage stage of fathead minnow eggs. Concentrations of 0.1, 1.0, and 5.0 mg/l of hydrazine for 24 hours resulted in a number of physiological defects compared to 0.01 mg/l and the controls. Hatching of fish exposed to 5.0 mg/l was significantly less than the controls and hatching was delayed in concentrations of 1.0 and 5.0 mg/l. Embryos exposed to 1.0 mg/l showed significantly different development than controls at 2 weeks. A 48-hour exposure at 5.0 mg/l was lethal and 1.0 mg/l caused severe deformities, while <1.0 mg/l did not adversely affect developing embryos. Pure linoleic acid hydroperoxide was incubated with carp intestinal extract or acetone powder and the decomposition rate of hydroperoxide and the formation of carbonyl compounds was investigated by Hata *et al.*³⁰⁴ In addition, the inhibitory effects of EDTA and cyanide were examined to measure the influence of metal ions and heme compounds on the enzyme reaction. Both systems decomposed approximately equivalent amounts of hydroperoxide and carbonyl compounds, unsaturated carbonyls were more elevated in experimental system than the control. EDTA and CN had only a partial inhibitory effect on the reaction.

Notopterus notopterus were exposed to four sublethal concentrations of phenol and PCP for 10, 20, and 30 days to study the effects on the hepatic acid and alkaline phosphatases and succinic dehydrogenase.³⁰⁵ The fish exposed to toxicants were found to have significant enzymatic differences in the liver compared to those found in the liver of control fish, with the activity of acid and alkaline phosphatase and succinic dehydrogenase inhibited in almost all cases. The greatest inhibition was observed in acid phosphatase after an exposure period of 30 days. In another study,³⁰⁶ fish exposed *in vivo* to phenol, DNP, PCP, and the synergistic combination of the compounds had a more pronounced stimulation of acid phosphatase. Stimulation of the alkaline phosphatase was greater for the antagonistic and additive combinations. Verma *et al.*³⁰⁷ found in other tests with these compounds and *Notopterus notopterus* that RBC, WBC, and the packed cell volume tended to increase while clotting time (CT), erythrocyte sedimentation rate, hemoglobin (Hb), mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, and mean cell volume (MCV) decreased when tested with a single chemical. However, PCP showed dual effects in many of the blood parameters. PCP caused CT, MCV, and Hb to decrease and DNP alone caused RBC to decrease. The additive chemical combination caused CT and MCV to decrease while the synergistic chemical combination resulted in an increase in CT and MCV. Hb increased in both additive and synergistic combinations. Histological examination of the livers of the bluegill sunfish exposed to sublethal concentration (0.1 mg/l) of PCP was investigated by Owen and Rosso.³⁰⁸ Cytological changes were similar for starved unexposed and PCP-exposed bluegills. Most of the degenerative changes, such as hepatocyte cord disorientation, multikaryotic cells, pyknotic nuclei, and eosinophilia, occurred after only 2 days of exposure with an overall improvement of increased cord orientation and decreased cellular effects seen by Day 4. The metabolism of ¹⁴C-phenol at sublethal concentrations in eight species of fish was studied

by Layiwola and Linnecar.³⁰⁹ Phenyl sulphate was found in all species while phenyl glucuronide was found only in the bream (*Abramis brama*), perch, roach, and the rudd (*Scardinius erythrophthalmus*). The effects of phenol (0.5 to 18 mg/l) on the predation of fingerling rainbow trout by adult rainbow trout was investigated by Schneider *et al.*³¹⁰ Fingerlings exposed to phenol concentrations at 7.0 mg/l to 10 mg/l were consumed at a significantly greater rate than the controls. Concentrations greater than 10 mg/l resulted in acute mortality.

Carp were fed PCB (Aroclor 1248) to observe the effects on the composition and enzyme activities of serum.³¹¹ The serum glucose content increased significantly after the PCB was ingested and an increased globulin content accounted for increased total protein content. The presence of Aroclor 1248 resulted in significant increases in the activities of various serum enzymes. A significant decrease in lysosomal and microsomal β -glucuronidase in the hepatopancreas was noted. Hendricks *et al.*³¹² studied the null effect of dietary Aroclor 1254 on hepatocellular carcinoma incidence in rainbow trout exposed to Aflatoxin-B₁ (AFB₁) as embryos. At 12 months, liver cancer incidence in combined AFB₁-treated, control diet groups, and the AFB₁-treated Aroclor 1254 diet groups were essentially the same (65.8 and 63.9%, respectively). Significantly higher liver to body weight ratios of Aroclor 1254 fed trout were seen at 12 months, but not at 9 months. Levels of accumulation of Aroclor 1254 on whole fish basis were 75 μ g/g at 12 months. The only tissue changes induced by Aroclor 1254 were glycogen depletion of hepatocytes and splenic hyperemia. Addison *et al.*³¹³ found that brook trout fed a single dose of Aroclor 1254 had elevated hepatic mixed-function oxidase activity shown by the ethoxycoumarin O-de-ethylase activity. In two dose-response experiments, the hepatic O-de-ethylase activity increased with increased PCB residue concentration from one feeding of Aroclor 1254. Ito *et al.*³¹⁴ studied the effects of PCB on the hepatopancreas lysosomal membranes of the carp. After a 14-day exposure to PCB, hydrolase, acid phosphatase, and β -glucuronidase were released in greater quantity than in the controls. Sodium copper chlorophyllin, α -tocopherol, lecithin, and cortisone inhibited the release of acid phosphatase and β -glucuronidase from the lysosome fraction of carp hepatopancreas *in vitro*. The effects of PCBs and β -naphthoflavone on the cytochrome(s) P450 and mixed-function oxidase activity in liver and kidney tissues of carp were investigated by Melancon *et al.*³¹⁵ Hepatic microsomal enzyme activity and P450 content were increased in carp injected with 200 mg/kg of Aroclor 1254 and 100 mg/kg β -naphthoflavone but not by the injection of 100 mg/kg of 2,4,2,4'-tetrachlorobiphenyl. Control and some induced hepatic microsomal enzyme activities were unaffected by metyrapone but were significantly inhibited by α -naphthoflavone *in vitro*. Kidney microsomal enzyme activities were generally unaffected by these agents. Förlin³¹⁶ investigated the effects of Clophen A50 (Cl-A50), 3-methylcholanthrene (3-MC), Pregnenolone-16 α -carbonitrile (PCN), and phenobarbital (PB) on the hepatic microsomal cytochrome P450-dependent monooxygenase system in rainbow trout. Only 3-MC and Cl-A50 markedly elevated the total amount of cytochrome P450 and the cytochrome P450-dependent metabolism of parnitroanisole and benzo(a)pyrene in fish liver. Förlin and Andersson³¹⁷ also studied the effects of Cl-A50 on the metabolism of parnitroan-

isole in an *in vitro* perfused rainbow trout liver. The main metabolites were paranitrophenol and paranitrophenol-glucuronide and the rate of paranitroanisol metabolism was markedly higher in perfused liver from Cl-A50-treated fish than untreated fish liver. The sublethal effects of dietary Cl-A50 on growth, reproduction and swimming performance in the minnow, *Phoxinus phoxinus*, was studied by Bengtsson.³¹⁸ Cl-A50 had a growth promoting effect and several effects on reproduction were noted. The minnows swimming performance was not affected at the concentrations that affected growth and reproduction.

Fathead minnow were exposed to phenanthrene adsorbed to <125 μm coal particles to determine bioaccumulation and changes in histology.³¹⁹ No obvious damage to external surfaces was seen, and particulates did not accumulate in internal tissue. However, ingestion of coal particles was observed and extensive mucous secretion by the gut mucosa followed. Mucous globules in coal-exposed epithelium was noted by electron micrographs of the gut but no erosion of the microvilli was seen. Elimination of coal from the gut and phenanthrene from the tissues was rapid, neither were detectable after a 24-hour depuration period. The effects of the anionic detergent, SLS on the vascular response to noradrenaline in isolated perfused eel gills was studied by Stagg *et al.*³²⁰ SLS was found to inhibit noradrenaline-induced vasodilation; the inhibition seemed to be noncompetitive and was reversible at low concentrations (6 $\mu\text{g/l}$).

INDUSTRIAL AND MUNICIPAL EFFLUENTS

Chlorine and wastewater. The effects of chlorinated wastewater in the Sheep River, Alberta, Canada were studied by Osborne *et al.*³²¹ It was found that fish moved in and out of a plume of chlorinated effluent. The effluent was toxic to rainbow trout (*Salmo gairdneri*) in 9 hours at the level of 0.92 mg/l total residual chlorine (TRC). Five species, including the rainbow trout, were found in the effluent plume. A study on the toxicity of the prechlorination effluent of 24 municipal activated sludge plants and five waste stabilization ponds was reported by Meitkosh *et al.*³²² Twenty-five percent of the secondary effluents sampled in the summer and 55% of those sampled in the winter were lethal to rainbow trout. Ammonia and anionic surfactants were suspected to be the cause of toxicity, although heavy metals were not measured. Field and laboratory avoidance of a heated and chlorinated power plant effluent by local fishes was examined by Giattina *et al.*³²³ Spottfin shiner (*Notropis spilopterus*) and whitetail shiner (*Notropis galacturus*) avoided laboratory concentrations between 0.19 to 0.28 mg TRC/l at 27° to 30°C and were less sensitive avoiding 0.23 to 0.43 mg TRC/l when temperatures fell from 26° to 7°C. Larval white perch (*Morone americana*) were subjected to chlorine and temperature stress after acclimation at 15° and 23°C.³²⁴ Mortality of larvae was generally greater at 23°C than at 15°C. Mortality of test organisms during a 4-hour exposure with animals acclimated to the highest temperature was greater than 85% at test temperatures greater than 6°C regardless of other treatment factors. In a similar study,³²⁵ eggs and larvae of striped bass (*Morone saxatilis*) were exposed to intermittent chlorine doses simulating effluent discharge conditions. Immediate mortality responses were cor-

related with chlorine concentration but latent mortality was correlated to exposure time, ΔT , life stage, and TRC. Chlorine levels tested were 0.15 and 0.30 mg/l.

The relative toxicity of hypochlorous acid (HOCl) and the hypochlorite (OCl^-) to mosquitofish (*Gambusia affinis*) was examined by Mattice *et al.*³²⁶ Mosquitofish were exposed for 1-hour periods to free residual chlorine at pH ranging from 6.05 to 8.42. Toxicities in terms of TRC varied inversely with pH ranging from 0.41 to 1.28 mg Cl_2/l , respectively. Hypochlorous acid was found to be about 4 times as toxic as OCl^- to this species. The same authors³²⁷ developed a system for short-term square wave concentration exposure of chlorine concentrations. A discussion of the equipment was followed by the results of exposures of *Daphnia magna* and mosquitofish to chlorine. The chlorine LC50 values for mosquitofish were 1.59 and 0.84 mg/l for 30- and 60-minute exposures, respectively.

Booth *et al.*³²⁸ studied the effects of intermittent chlorination on plasma proteins of rainbow trout. Greatest alterations in blood protein composition was noted in fish exposed to 0.22 mg/l which also appeared to be the lethal limit for this fish. In addition, chlorine concentrations of 0.11 and 0.22 mg/l increased the cough frequency of trout after 1 hour of exposure. Greater increases in coughing occurred in fish exposed to 0.22 mg/l. The physiological responses of rainbow trout to intermittent monochloramine exposure was observed by Travis and Heath.³²⁹ Trout responded to chloramine pulses of 0.4 to 0.6 mg/l with increases in opercular movement, bradycardia, and cough rate. Other blood parameters such as arterial PO_2 and methemoglobin were altered but little, if any, hypoxemia resulted. Grizzle *et al.*³³⁰ reported on the high incidence of oral papillomas in the black bullhead (*Ictalurus melas*) which were living in a wastewater treatment pond containing 0.2 mg/l residual chlorine. No lesions were found on green sunfish (*Lepomis cyanellus*) and golden shiners (*Notemigonus crysoleucas*) collected from the same pond.

The effects of pollutants associated with the coal industry in South Wales and particularly coal solids from a mine on fish populations were described by Scullion and Edwards.³³¹ Brown trout (*Salmo trutta*) occurred at low densities (<0.05/m²) downstream from acid and ferruginous drainage, in the latter case caused by impoverished food supplies rather than from direct toxic effects. High egg mortalities (>80%) occurred in reaches receiving ferruginous and suspended solid wastes. Riggs and Gregory³³² studied the effects of the Decker Mine, the largest coal mine in the western U. S., on the walleye (*Stizostedion vitreum*) and sauger (*Stizostedion canadense*) population in the Tongue River Reservoir in southeastern Montana. Analyses of 645 walleyes and 546 saugers indicated that water effluents from the mine, under present conditions, had no significant effect on these populations. Penkal and Gregory³³³ evaluated the potential effects of a surface coal mining operation on Montana's Tongue Reservoir. Smallmouth bass (*Micropterus dolomieu*) and largemouth bass (*Micropterus salmoides*) were not affected to any great degree by the mining operation. The only effect noted was a general correlation between reproductive success and turbidity. Leachate toxicity derived from the solid wastes of coal gasification and coal liquefaction were studied by Suloway *et al.*³³⁴ Leachate derived from coal liquefaction solids were toxic at dilution of 10 to 0.005%, depending on the process solid used. The test

animals were 1- to 5-day-old fathead minnows (*Pimephales promelas*) in static conditions. Turnpenny and Williams³³⁵ examined the factors affecting the recovery of fish populations from the Ebbw Fawr River in a South Wales (G. B.) coalfield area polluted by wastewater and wastes from coal, iron or steel, and associated industries. Reduced levels of toxic materials, restored DO levels and the reoccurrence of acceptable pH levels from pollution control measures resulted in the recolonization of some fish species in this river. It was suggested that a sport fishery could be developed in the near future provided appropriate management practices were adopted. The impact of surface coal mine reclamation on headwater streams in southwest Virginia were described by Matter and Ney.³³⁶ Alkalinity, hardness, sulfate, suspended solids, and fine particle sediment were higher in the mining-impacted streams, while pH was lower. Benthic invertebrates and fish were more abundant in reclaimed streams. Continual sedimentation as a result of surface mining was emphasized as being an especially serious concern. Goettl and Edde³³⁷ reported on an investigation of the Trout Creek in Colorado concerning the possible effects of strip mine drainage on the fish fauna. The authors reported no changes attributable to such pollution on this stream. Magnuson *et al.*³³⁸ studied the population dynamics of wetland receiving effluent from a coal-fired electric generating station adjacent to the Wisconsin River. The authors reported no observable effect from the power plant on fish-spawning use of the swamp marsh although metal concentrations in bluegill sunfish (*Lepomis macrochirus*) were found to be elevated. The effect of pollutants on an Indian river system was studied by Mhatre *et al.*³³⁹ Heavy metals appeared to exert the most detrimental effect on the river although other contaminants were present. Bull *et al.*³⁴⁰ analyzed roach (*Rutilus rutilus*), perch (*Perca fluviatilis*), and pike (*Esox lucius*) reared in wastewater lagoons for Hg content. Levels of 150 to 350 ng/g were found in such fish with much of the Hg being methylmercury.

Elizabeth *et al.*³⁴¹ studied the lymphoid cells of the head kidney of *Rasbora daniconius* under stress from fertilizer factory effluents. Ammonia, phosphoric acid, and sulphuric acid were the main constituents. All effluent types affected the lymphoid cell after 24-hour exposures but a different group of cells were affected by ammonia rather than by the two acid effluents. The effects of distillery wastes on the metabolism of several species of fish from India were reported by Shaffi.³⁴² An inverse relationship between concentration of distillery waste (5, 10, 15, and 20 mg/l) and liver and muscle glycogen depletion was reported. Serum glucose and lactate levels increased with increased industrial effluent concentration.

Pulp and paper. The effects of exposure of a treated bleached kraft mill effluent on green sunfish, golden shiners, hybrid bream (*Lepomis macrochirus* × *Lepomis microlophus*), channel catfish (*Ictalurus punctatus*), and red swamp crayfish (*Procambarus clarki*) were evaluated by Graves *et al.*³⁴³ No toxicity attributable to the effluent was found on any of the tested animals in 20- to 30-day continuous-flow tests. The authors concluded that a final stage treatment employing an aerated stabilization basin (ASB) was adequate to prevent deleterious toxic effects of this type of effluent to the aquatic environment. The acute lethal toxicity of waste waters from a Finnish kraft pulp mill to rainbow trout was determined by Holmbom and Lehtinen.³⁴⁴ Debarking waters were the most

toxic on a volume basis giving a 96-hour LC50 value of approximately 1.5% vol/vol. Bleaching wastewaters resulted in the highest toxicity of the waters studied. The results indicated that it was feasible to predict toxicity by calculating toxic unit values from analytical determinations of toxic compound concentrations. Lehtinen and Oikari³⁴⁵ used a swimming test to detect sublethal effects of kraft pulp effluent to European perch at 14-day exposures to 2% effluent, reduction of swimming ability occurred, when compared controls in dilution water. Leach and Chung³⁴⁶ developed a chemical analysis procedure to rapidly measure compounds responsible for the toxicity of pulp mill effluents to fish. The results were converted via toxic units into estimates of acute lethal toxicity and to determine loadings into river systems. Agreement between 96-hour LC50 values calculated by this procedure and values from bioassays with rainbow trout were within 30% for 73% of the samples examined. Estimates based on theoretical dilution capacity of receiving waters showed that secondary treatment was beneficial in protecting rivers against toxic effects of pulp mill effluents. Tomlinson³⁴⁷ compared a series of tests with fish by two laboratories in Canada to examine individual process stage effluent streams from a pulp mill for the presence of toxicants and to establish any interactions that may occur in the effluent system enroute to their entry into receiving waters. Toxic units for several effluent components were compared and evaluated. The effects of different types of toxicants on the toxic contribution of pulp mill effluents were also examined.

MISCELLANEOUS

The mutational changes in the courtship activity of male guppies (*Poecilia reticulata*) after X-irradiation was examined by Werner and Schröder.³⁴⁸ Results showed that 1 000 R decreased courtship activity and increased aggressiveness. Higuchi *et al.*³⁴⁹ reported on food chain and direct water uptake of tritium. The uptake directly from water was always less than unity. Concentration effects through the food chain including bacteria, diatoms, brine shrimp, and fish (*O. latipes*) were found. Biological half-lives of tritium in organisms labelled through the food chain were considerably longer than that from those labelled by tritiated water alone. The effects of the Three Mile nuclear accident on the fishery of the Susquehanna River were examined by Hickey.³⁵⁰ The report showed that the harvest index was lowered for 6 months subsequent to the accident but all of the change was the result of less fishing pressure and release of caught fish rather than direct biological effects from radiation.

Sigler³⁵¹ described the effects of chronic turbidity on feeding, growth, and social behavior of steelhead trout (*Salmo gairdneri*) and coho salmon (*Oncorhynchus kisutch*). Laboratory fish reared in clear water were larger and increased in weight and length more rapidly than fish in turbid water. Larger numbers of fish remained in clear water channels after the initial growth phase. Reticulate sculpins (*Cottus perplexus*) were monitored for 7 years before and 7 years following clear-cut logging in a tributary of the Alsea River in Oregon.³⁵² Environmental changes such as low DO, increased turbidity, and other physical alterations caused by logging caused some decreases in population in some tributaries, although the size of the fish generally increased after logging. The influence of a reservoir discharge on fish health below a

reservoir in Georgia was examined by Grizzle.³⁵³ The effects of metal accumulation, pH, and DO on toxicity and physiological responses of four species of fish were discussed.

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